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An Address.[1]

By G. L. BENNETT,

President of the South Australian Branch of the British Medical Association.

LET me say first of all how much I appreciate the honour of having been your president for the last twelve months; it is an honour the memory of which will remain with me for years, and I can only hope that I have been worthy of it.

If a title is necessary for my address tonight, I would suggest that it be "Some Thoughts on General Practice of Yesterday, Today and Tomorrow".

At the outset I should like to acknowledge the help I have received from that most entertaining and instructive book written by Stephen Taylor entitled "Good General Practice". Stephen Taylor received a grant from the Nuffield Foundation to conduct a non-statistical survey of a number of practices of general practitioners, recognized as good doctors by their colleagues, to see how far the lessons they had to teach could be generally applied. When I knowingly quote from this work due acknowledgement will be made; but it may be that I unknowingly put forward some views as my own which have perhaps been influenced by Stephen Taylor. I certainly commend this book to all of you.

[1] Read at the annual meeting of the South Australian Branch of the British Medical Association on June 27, 1956.

Definition of a General Practitioner.

Can we define what is meant by a general practitioner? Many different definitions have been given, and perhaps it is hard to make one definition for the whole picture. However, I strongly favour the term "family doctor"—a term which is being drilled into the school-children in their health broadcasts by one of our eminent members. I think that in Australian conditions at least this is the term which most aptly describes a general practitioner, and a term that I would like to see kept in the public mind.

General Practice of Yesterday.

Now let us look at general practice of yesterday—and by "yesterday" I mean the time between the two World Wars. From my own observation and from discussion with my senior colleagues, it seems that general practice varied very little over that period. For the most part fees remained stable, as did to a large extent the cost of living, of motor-cars, and of instruments, income tax *et cetera*.

The general practitioner was "father confessor" to his patients, and held their respect and trust, and in most cases their devotion. The lodges were very active, and most general practitioners had a large lodge practice which was worked on a capitation service, and a very good service indeed was given. The general practitioner took a lead in

public affairs, particularly so in the country. In this period, too, it was common for specialists to graduate from the ranks of general practice, and it was far from uncommon to see the part-time specialist, part-time general practitioner. Nursing and hospital facilities appeared much easier to obtain. All in all, I believe that the doctor-patient relationship was on a high level.

General Practice Today.

Now let us look at the changes which have taken place from the end of World War II until today. I believe that several years' organized service under military control by large numbers of the younger practitioners made the subsequent changes much easier of implementation.

Probably the one big factor which has led to many of the changes is the increased cost of living, which still continues.

The first big change was brought about in this way when a new lodge agreement was proposed. After lengthy negotiation, the old lodge system with its capitation fee and individual doctors' lists gave way to a fee-for-service system and no definite doctors' lists.

This was followed by the gradual evolution of the *National Health Act*, with its three offshoots—(i) the *Pharmaceutical Benefits Act*, (ii) the Pensioner Medical Service and (iii) the medical insurance scheme.

After some early teething troubles this scheme appears now to be working well, from the point of view of both the patient and the doctor. It is a scheme which more than bears comparison with any other health scheme throughout the world, and leaves the doctor much freer of bureaucratic control than most others. However, if we wish to keep this freedom it is up to the medical profession itself to see that the doings of a small minority are not allowed to tar the whole profession with the same brush. We must take the lead and exercise control of those members who tend to bring the profession as a whole into disrepute.

Being a member of the medical services committee of inquiry for this State, I feel very strongly on this matter, as I have seen and endeavoured to correct many of the abuses taking place. It is far better for the profession itself to stamp out these abuses than to have further government control with the ensuing penal clauses.

In any fee-for-service scheme the practitioner must work conscientiously, or else the old capitation scheme, which needs far less government checking, will return.

A short while ago I was a guest at the annual dinner of a kindred association, and when I was introduced to the Premier, he said: "Tell me, Doctor, are you a profession or a union?" I quickly answered: "A profession, sir." However, I have often wondered if some of our members are not forgetting the high ideals of our profession and considering it more as a business. Are not some of us becoming too "money-hungry"? It is true that many inroads are being made into our profession; but are we considering those inroads on an idealistic basis, or on the basis of how they will affect our pockets? I put that forward only as a thought for your consideration.

A further advance has taken place in the changes in the *Workers' Compensation Act*.

Many of us remember the days when a worker received only a pittance when he was off duty through injury. This absence sometimes extended over some weeks or months, during which time medical attention was needed. However, with such a small income, the injured worker was unable to pay our just fee, so that a large percentage of this work was done by the profession in an honorary capacity. Nowadays we can look to the employer, through his insurance company, for payment for this very necessary service. This, I feel sure, has led to a better relationship between the patient and the profession, giving the patient a feeling of independence, and the doctor the assurance of payment for work done.

The great advances in medical science over the last ten to fifteen years have meant that the general practitioner is now able to do more and more for his patient. Even a poor general practitioner nowadays can often do more for his patient than the best specialist of years gone by. I refer to such things as pneumonia, child-bed fever, meningitis *et cetera*. However, do not let us forget, in these days of improving medical remedies, the lesson learnt by our earlier practitioners in the hard school—that the comfort and peace of mind of the patient should still be in the forefront of our minds. A sick patient still needs comfort, sympathy and good nursing, as well as modern antibiotics.

Another change which appears to have been unavoidable, but which nevertheless I am sorry to see, is the difficulty nowadays of graduating through general practice to specialism. Few would disagree with me when I say the ideal specialist is one who has dealt with the patient as a whole as the family doctor, before later concentrating on one special branch of medicine. However, advancing knowledge with its attendant long years of preparation and study makes it extremely hard for a surgeon, for example, to have five years or so in general practice before commencing to specialize. This is a matter for further consideration for those who plan our education at the post-graduate level.

General Practice Tomorrow.

Let us now pass on to some suggestions for the future. As Stephen Taylor states:

> It is a fundamental conclusion that good general practice begins with the good general practitioner. The future of general practice is in the hands of the general practitioners themselves. They are in a position to provide a unique service, because they alone can offer the patient and his family continuity of medical care over the years. They alone can know family environment. And they alone can learn what is the normal for each individual patient.
>
> Provided that the general practitioners use to the full their unique opportunities for true clinical medicine, they are in no danger of disappearing from the medical scene.
>
> If, however, they regard themselves only as signposts on the way to the specialists, and conduct their practices accordingly, then the case against general practice becomes a strong one.
>
> If there is a single unmistakable characteristic of general practice, it is to be found in the continuing observation and care of the patient from month to month, from year to year, and from illness to illness. It is this continuity of care which enables the general practitioner to make his unique contribution to medicine

Good General Practitioner Needs the Ability of Good Specialist.

Here and now I would like to say that it is my view that a good general practitioner is not a general practitioner only because he has not the ability to become a specialist. General practice is only one branch of our profession, and its efficient conduct calls for the very best in man. A sense of duty and vocation is a most necessary asset. Good physical health and unbounded energy are required to conduct a practice efficiently. And here, I think, is the necessity for more relaxation by group practice or roster system. The roster system may be within a partnership, or arranged amongst a group of general practitioners in the locality. If we are to hold the trust and confidence of the community, it is necessary for us to give a full-time service.

Some time ago I was speaking to a fellow general practitioner at a football match, and said: "What do you do when you come away for the afternoon—do you have a roster system?" He said: "No! There are plenty of doctors in the city these days, and if my patients can't get me, they can call someone else." That, I believe, is wrong both in principle and in practice. It leaves his "out of hours work" to be done by his fellow practitioners without any means of reciprocation, and only leads to ill-feeling amongst his fellow practitioners and his patients.

I quite agree that with the vast increase in work of general practice nowadays it is quite impossible for a man to give a continual twenty-four-hour service himself.

Therefore I make a plea for further extension of the roster system; this allows one to have relaxation, to have time off for meetings and medica lreading, to have more family life. More important still, it allows the general practitioner to maintain his good general health and to be of more value to his patients. In other words, it improves not only the welfare of the doctor but also the welfare of the patients.

However, a corollary of good group or partnership practice is the keeping of good case notes and records. The day when the general practitioner felt he could carry everything in his head has disappeared. It is only by good case notes of previous illnesses that we can reap the full value of continuity of treatment of the patient. They are also necessary when one's partner or roster colleague has to treat the patient in emergency in one's absence. Far from wasting time by keeping good records, I have proved to my own satisfaction that this is a great time-saver. We also owe it to our patients, who may at a future date need copies of these records in their claims for assistance in repatriation, workers' compensation cases, life assurance *et cetera*.

Medical Education.

Now I should like to make a few remarks on medical education, both at the undergraduate and at the post-graduate level.

A big step forward has been taken during the last two years in alloting fifth year students to general practitioners for a few weeks during their Christmas vacation. This has been advocated by many of us for some years. As far as I can see, it is working very well. I should like to go a step further, and suggest that perhaps one or two ward rounds could be conducted by general practitioners in which they could explain to students how these patients were examined outside and how they were dealt with. I think also that two or three lectures on general practice during the course would be of assistance for the future.

At the post-graduate level, more and more facilities are becoming available for the general practitioner who wishes to keep up to date with modern advances. The Post-Graduate Committee in Medicine runs regular refresher courses. I would suggest that in all partnership agreements a clause be inserted arranging for so much time off each year to attend some of these courses. Lecturers are also sent to country districts for week-end courses, and these are making a great contribution. This year clinico-pathological demonstrations are being held during the lunch hour at advertised times. I would commend these to your notice, and should like to see the scheme extended further.

The British Medical Association holds a scientific meeting on the last Thursday of each month. An attempt is made to choose subjects which will interest the general practitioner. In spite of this attendances are sometimes poor. Medico-political meetings, at which the subject perhaps affects one's pocket, draw a very much larger attendance.

The College of General Practitioners.

As many of you know, the College of General Practitioners was formed in England some years ago, and some Faculties are now in existence in some States in Australia. It is not a medico-political body—that is left to the British Medical Association; but its aims are to improve the education of the general practitioner and to aid in research on the general practice side.

Dr. Talbot Rogers, who recently came to Australia as a representative of the Parent Body of the British Medical Association to present the Gold Medal of this Association to Sir Henry Newland, is an active member of the College of General Practitioners in England. I was fortunate enough to be able to discuss this matter with him. Although I am in whole-hearted agreement with its aims, he agreed with me that at present in South Australia the Post-Graduate Committee in Medicine and the British Medical Association were endeavouring to carry out the aims of the College in this State, and perhaps until our membership became larger, we could well carry on as we were. However, I bring the matter up for your thoughtful attention.

Resident Medical Officers Undertaking Apprenticeship in General Practice.

Another interesting idea has been in operation for some years at the Royal Newcastle Hospital. The hospital bought a share in an old-established general practice, and resident medical officers, as part of their two-year tour of duty, spend a certain time working this general practice under the watchful eye of the senior practitioner.

I believe that this idea is worthy of consideration here. If it should ever happen that we have the number of graduates available, I think that three to four weeks of their roster could be spent acting as *locum tenentes* in a well-established partnership practice. This would serve the dual purpose of allowing one of the principals to have a holiday and at the same time fitting the young graduate for future general practice.

Local Groups and Associations.

A further suggestion I should like to make is the formation of more and more local groups and associations within our Branch of the British Medical Association. This not only brings members together at regular intervals and leads to more goodwill among neighbouring practitioners, but is a further step in post-graduate education. It also encourages more interest in the affairs of the Branch, and we have had many very active members of Council who have initially become interested in Branch affairs through their local group.

Here I should like to emphasize what Dr. Edgar Thomson, President of the New South Wales Branch, recently said—namely, that one should not join the British Medical Association just to be on the receiving end. One must ask onself what one can give for the welfare of the Association.

Plea for Unity Between General Practitioner and Specialist.

Let me at this stage make a plea for complete unity between the general practitioner and the specialist. We are all members of the one great profession, and for the honour of the profession alone, unity is essential.

Possibly the greatest rift which has occurred has been due to workers' compensation cases. It is not a serious rift, and I am confident that it can be healed if each group would put itself in the place of the other and follow the old golden rule. Combined meetings of both groups, in which minor differences can be discussed and ironed out, are in my opinion of great advantage to all concerned.

Conclusion.

And now, although there are many other things that I might say, I should like to leave you with this thought, well expressed by Stephen Taylor:

> By their standards of work and by their conduct the General Practitioners must deserve to be free. This is the challenge of general practice today. The future is in the Doctors' own hands.

THE AUSTRALIAN RED BACK SPIDER (LATRODECTUS HASSELTII): II. EFFECT OF TEMPERATURE ON THE TOXICITY OF VENOM.

By SAUL WIENER, PH.D., M.B., B.S.,
Commonwealth Serum Laboratories, Melbourne.

DURING routine assays of the venom of the red back spider (*Latrodectus hasseltii*) in mice, it was observed that on hot days the animals died earlier and the average lethal dose of venom was smaller than on cold days.

Further study of the effect of external temperature on the toxicity of venom appeared desirable for the following two reasons. Firstly, there is a popular belief in Australia that a bite by *L. hasseltii* is more dangerous in summer than in winter. Bücherl (1953), referring to a poisonous spider of South America, quotes the views held by older investigators that in hot seasons spiders are more venomous than in cold seasons. Secondly, when the potency of an antivenene is determined by biological assay, it is advisable, for purposes of accuracy, to use many lethal doses in each of the mixtures of venom and antivenene that are injected into an animal. In the case of antitoxin, it has even been postulated that it should be titrated by the amount necessary to protect an animal against the largest number of lethal doses it is possible to inject (Trevan, 1927). In practice, because of the limited supply of venom, even where sera of high potency are available, the amount of venom which is used for assaying antivenene is the minimum amount required for obtaining a reasonably reliable estimate of the neutralizing capacity of the serum. Thus, for assaying tiger-snake antivenene, an amount of venom corresponding to 25 times the LD50 dose is used at the Commonwealth Serum Laboratories (Graydon, 1956). In the case of red back spider venom, because of its scarcity, only four LD50 have been used for assaying antivenene (Wiener, 1956).

If the amount of spider venom which represents one LD50 dose can be reduced appreciably in any way, it will result in a saving of venom when antivenene is assayed. At the same time, it will become practicable to increase the number of lethal doses of venom in each dilution of antivenene when the latter is assayed.

In the present communication, data are presented to show that the toxicity of spider venom in mice can be increased considerably by altering the temperature of the environment in which the animals are kept.

Methods and Materials.

The method used for obtaining venom from the dissected venom glands of *L. hasseltii* and the method of preparation of antivenene have been described previously (Wiener, 1956). A stock solution containing the venom of one spider per millilitre was prepared. The amount of venom injected into an animal was expressed in fractions of a spider. Thus, one millilitre of a 1:10 dilution of the stock solution corresponded to 0·1 spider.

All injections were made intraperitoneally into male mice weighing between 22 and 30 grammes. The mice were placed in a cold room at 0° C., in a warm room at 37° C. and in an ordinary room, the temperature of which varied between 18° and 25° C. The animals were kept at these respective temperatures for twenty-four hours prior to being injected with venom, and afterwards were again observed at these temperatures for three to four days.

Effect of High and Low Temperatures on Mice.

If mice are kept for any length of time at an external temperature of 37° C., ample ventilation must be provided. An electric fan was used and only six mice were placed in each cage, which measured 14 inches by 10 inches by four inches. In addition, plenty of drinking water was supplied. However, because not infrequently some mice died as a result of exposure to a temperature of 37° C., later experiments not reported here but yielding similar results were carried out at 35° C. At either temperature, after an initial period of restlessness, the animals were lying stretched out and separated from each other on the floor of their cage. Their mouths were slightly opened and respiration was shallow and rapid. Their appetite was poor and there was loss of weight. If they were disturbed, the animals showed signs of hyperexcitability. The mean rectal temperature of mice which have been kept for twenty-four hours or more at 37° C. is shown in Table I. In one instance the rectal temperature of a mouse which was comatose and died a few minutes later was 108·6° F.

The animals had no difficulty in adapting themselves to an external temperature of 0° C. Their fur became ruffled and the animals huddled together. The mean rectal temperature of mice after twenty-four hours at 0° C. and at room temperature is shown in Table I.

The rectal temperature of mice was recorded with a specially designed resistance thermometer ("Thermister").[1] The tip of the instrument was inserted 25 millimetres past the anal sphincter. At room temperature the difference in rectal temperature when the instrument was inserted five millimetres and 25 millimetres amounted to 2° F., whilst at 0° C. a difference of as much as 5° F. could be observed. A small rubber ring, placed at a distance of 25 millimetres from the tip of the instrument, ensured proper depth of insertion as well as acting as a seal when pressed against the anal sphincter. A similar device has been used by Sumner (1913), who also observed variations in rectal temperature with depth of insertion of the thermometer.

TABLE I.

Body Temperature of Male Mice at Different External Temperatures.

External Temperature.	Number of Mice.	Mean Rectal Temperature.	Standard Deviation.	Standard Error of Mean.
0°C	70	95·39° F.	4·01° F.	0·48° F.
18° to 24° C.	80	101·21° F.	0·91° F.	0·10° F.
37° C.	60	104·30° F.	0·99° F.	0·13° F.

Toxicity of Venom at Various Temperatures.

The results of one of many experiments designed to show the effect of spider venom in mice at three levels of external temperature are shown in Table II.

TABLE II.

Toxicity of Venom at Various External Temperatures.

Venom Fraction of One Spider.	18° to 25° C.		37° C.		0° C.	
	Number of Mice.	Number which Died.	Number of Mice.	Number which Died.	Number of Mice.	Number which Died.
0·4	7	7	—	—	—	—
0·3	7	7	—	—	—	—
0·2	7	6	—	—	—	—
0·1	7	3	—	—	—	—
0·05	7	1	—	—	—	—
0·02	7	0	7	7	7	7
0·01	7	0	7	7	7	7
0·005	—	—	7	7	7	7
0·002	—	—	7	4	7	6
0·001	—	—	7	0	7	2
0·0005	—	—	7	0	7	0
0·0002	—	—	7	0	7	0

It can be seen that both at 37° and at 0° C. the amount of venom necessary to kill 50% of mice corresponded to 0·2% of the amount of venom contained in one spider, whilst at room temperature the LD50 was 20% of the amount of venom of one spider. Thus at 0° and 37° C. the toxicity of venom was 100 times greater than at room temperature.

At 37° C. the mice died earlier than at 20° C. At the former temperature some of the animals that had received the higher concentrations of venom were already dead ninety minutes after the injection, and about 50% of the animals which died had done so four hours after the injection, the remainder being dead the next day.

A few of the mice kept at 0° C. which had received the higher concentrations of venom were dead four hours after the injection; the remaining animals which did not survive were dead within twenty-four hours.

None of the mice kept at room temperature died earlier than eight hours after the injection with any of the concentrations of venom indicated in Table II. With the higher concentrations of venom some were dead the next day, and

[1] Constructed by Bayliss Electronics.

others showed signs of paralysis and closure of eyes, deaths occurring over a period of four to five days.

Because of the relative speed with which the animals died at temperatures of 37° and 0° C., some of the signs of intoxication which frequently precede death at room temperature were not discernible. For instance, in none of the animals kept at 37° or 0° C. could any paralysis be observed. However, the normal depressing effect of spider venom intoxication on body temperature was greatly accentuated in an external environment of 0° C. Table III shows

TABLE III.

Effect of Lethal Doses of Spider Venom on Body Temperature.[1]

External Temperature.	Rectal Temperature.	
	Before Injection.	Three Hours After Injection.
0° C.	96·0° F.	70·5° F.
23° C.	101·4° F.	89·0° F.
37° C.	104·6° F.	99·0° F.

[1] Mean of five mice per group.

how body temperature was depressed three hours after the injection of lethal doses of venom at three levels of external temperature. Because of the consistency of this phenomenon, the eventual death of an animal could be predicted fairly reliably on the basis of the observed reduction in body temperature.

The absence of prolonged illness after the injection of venom when mice were kept at 0° and 37° C. considerably shortened the period during which the animals had to be observed when venom was assayed.

Mr. N. Semple of these laboratories has conducted preliminary experiments with tiger-snake venom on mice at different external temperatures. He has noticed a slight increase in toxicity at 0° and 37° C. compared with that

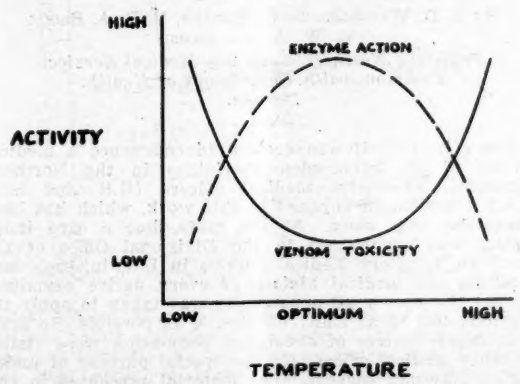

FIGURE I.

Diagram of the effect of body temperature on the toxicity of spider venom and the hypothetical rate of enzymatic destruction of venom.

observed at room temperature. At 0° C., the animals were either dead or had fully recovered twenty-four hours after the injection of various concentrations of tiger-snake venom. At either extreme of temperature the animals did not show paralysis or other signs of tiger-snake venom intoxication, which commonly occurs at room temperature with low doses of venom and may be followed by death several days after the injection.

The increased toxicity of spider venom at 0° and 37° C. has been utilized successfully for the titration of antivenene, with a considerable increase in the LD50 dose which was utilized and a more than tenfold saving of the amount of venom which had hitherto been necessary when the assay was carried out at room temperature. This will form the subject of a subsequent communication.

Effect of Acidosis and Alkalosis on the Toxicity of Venom.

Maretic and Stannic (1954) quote a statement of Vellard that the venom of Latrodectus becomes alkaline and highly toxic at temperatures above 25° C., whilst at lower temperatures it becomes acid and less toxic. In view of this and the reported effect of high external temperature on the alkali reserve of the body (Sundstroem, 1927), the following experiment was performed.

Two groups of mice received in their drinking water ammonium chloride (0·1%) and sodium bicarbonate (0·1%) for a period of fourteen days. At the end of this period the urine of mice which were receiving sodium bicarbonate was alkaline to litmus paper, whilst that of mice receiving ammonium chloride was acid. A third group of control mice receiving tap water had urine which was neutral or acid to litmus paper. In each of the groups of mice venom was assayed at 37° C., at 0° C. and at room temperature as described previously. At each level of external temperature there was no difference in the toxicity of venom whether the mice had been receiving sodium bicarbonate, ammonium chloride, or plain tap water.

In another experiment freshly prepared venom was placed at 4° C. and at 37° C. for a period of two hours. When both lots of venom were assayed in mice at room temperature, no significant difference in toxicity was observed. Storage of venom for twenty-four hours at 37° C. resulted in a loss of about 80% of its original toxicity. At 4° C. venom remained fully potent for a period of at least four days.

Discussion.

There are several references in the literature concerning the effect of external temperature on the toxicity of venoms and bacterial toxins.

Micheel and Jung (1936) state that when cobra venom is being assayed in mice it is important to keep the external temperature between 23° and 24° C. and that with lower external temperature the lethal dose is greater.

Schöttler (1955), in an exhaustive analysis of the variations in results when snake venoms and antivenenes are assayed, refers to the possible effect which external temperature may have on the resistance of small animals. He quotes a suggestion made by Slotta and Szyszka that higher environmental temperatures enhance toxicity. Schöttler's own data did not reveal any relationship between toxicity and external temperature, which in his experiments ranged from 10° to 30° C.

Grasset and Zoutendyk (1931) found that in certain lizards (Zonuridae) the time of death after the injection of tetanus toxin was shortened with rise of external temperature. The ratio of the times of death of lizards kept at 6°, 25° and 37° C. was 10:2:1 respectively.

More recently, Ipsen (1951), using groups of mice from the same litters, found that with amounts of tetanus toxin well beyond one minimum lethal dose survival time was shortened with increase in external temperature. Ipsen postulates that environmental temperature exerts its effect presumably through change in body temperature.

It is interesting to speculate why, in the case of spider venom, toxicity should be so greatly increased both at high and at low temperatures. The body temperature of mice certainly varies with the temperature of the environment. The difference in rectal temperature when mice are kept at 0° and 37° C. may amount to 8° or 10° F. This increase in body temperature at 37° C., and its decrease at 0° C., may affect either the toxicity of the venom itself or the tolerance of the animal's tissues to the action of the venom.

As venom deteriorates at 37° C., which is slightly below the average body temperature of mice, and in view of the fact that toxicity increased when the normal body temperature was raised or lowered a few degrees Centigrade, it is unlikely that the toxicity of the venom itself was affected.

It is more probable that increase and decrease of body temperature lowers the animal's resistance to the action of spider venom. Under normal conditions the mouse can cope with a small but definite amount of spider venom, which either causes no illness at all or produces toxic effects from which the animal recovers. It is this ability of the tissues to tolerate sublethal doses of venom which determines its minimum lethal dose. According to Banic and Lyubetic (1938), three-quarters of one certainly lethal dose is neutralized by the natural defences of the animal's organism. Any interference with the natural detoxifying mechanism of the body will reduce the lethal dose of venom. The observation recorded above, that at 0° and 37° C. mice either recover or die without signs of intoxication, further strengthens the belief that the effect of temperature is on the host rather than on the toxic agent.

The mechanism whereby the host's resistance to the effect of venom is decreased with raising and lowering of body temperature offers a fruitful field for speculation. Neither reducing nor increasing the alkali reserve of the body appears to play any part.

One hypothesis which may be entertained envisages that normally the body detoxifies venom through the action of enzymes, which are optimally active at normal body temperature. If body temperature is raised or lowered, these enzymes work less efficiently than at normal body temperature, with the result that less venom is detoxified. A diagram of such a mechanism is shown in Figure I. Support of the concept that changes in body temperature interfere with enzyme action comes from the experiments of Ito (1916), who has shown that in rabbits the synthesis of ethereal sulphates was reduced when the normal body temperature of the animal was either raised or lowered.

From a practical point of view, whatever the mechanism may be, the decreased tolerance of mice to spider venom at 0° and 37° C. can be utilized with advantage for assaying spider venom and antivenene. The adoption of such a method results in a considerable saving of the amount of venom which has to be used. At the same time, the period during which the animals have to be observed before results are recorded is shortened. For record purposes it becomes important to specify the temperature of the environment at which an assay has been performed.

With regard to the cause of the alleged greater toxicity of spider bites in summer, little is known. Bücherl (1953) could find no difference in the toxicity of the venom of a South American spider whether the venom was collected in summer or winter. However, he found that the amount of venom per spider which was obtained by letting the spider bite through thin rubber tubing was appreciably greater in summer than in winter. He relates greater toxicity to the larger amount of venom which may be injected when the spider bites in summer.

In the case of the red back spider, during the months from August to February, the amount of venom obtained by freeze-drying extracts of venom glands from 300 spiders has generally varied between 0·08 and 0·1 milligramme. However, in one batch of venom prepared from 40 spiders in January, the amount of venom which was obtained corresponded to 0·32 milligramme per spider. Thus, in summer, some spiders may have considerably more than 0·1 milligramme of venom. Whether or not similar large amounts of venom may occasionally be obtained from spiders during winter is not known.

Another factor which could contribute to the greater danger of spider bites in summer than in winter relates to the increased vascularity of the skin during hot weather. Under such conditions the rate of absorption of venom after a bite may be increased, with a corresponding reduction of the effectiveness of a tourniquet and other local measures which are used for the first-aid treatment of spider bites.

Summary.

1. The body temperature of mice varies with that of the external environment.

2. At 0° and 37° C. the toxicity of the venom of the red back spider is 100 times greater than at 18° to 24° C.

3. The possible causes of this effect are discussed, and its application for the assay of spider venom and antivenene is suggested.

Acknowledgement.

I am grateful to Dr. J. J. Graydon for calculating the data embodied in Table I.

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MANTOUX SURVEY OF FULL-BLOODED ABORIGINES IN THE NORTHERN TERRITORY.

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EARLY in 1951 it was decided to commence a medical survey of all full-blooded aborigines in the Northern Territory. Successive medical officers (R.B. and later W.A.L.) have been engaged in this work, which has been proceeding ever since. At the same time a card index system was commenced in the Divisional Office of the Northern Territory Medical Service in Darwin, each card detailing the medical history of every native examined. During the survey an opportunity was taken to apply the Mantoux test to as many aborigines as possible. In addition, many centres of aboriginal population were visited by other medical officers for the special purpose of undertaking Mantoux surveys. The material presented in this paper represents the combined results of all these efforts.

Population and Distribution.

The exact population of full-blooded aborigines is not known; but it is estimated, as a result of many surveys both by the Department of Welfare (formerly Native Affairs) and the Department of Health, that there are about 13,000 to 14,000 full-blooded aborigines. The approximate distribution of these is shown in Figure I. These aborigines today are living (*a*) around towns such as Darwin and Alice Springs, (*b*) on Government settlements, (*c*) on Church missions, (*d*) on cattle stations, and (*e*) as nomads in country uninhabited by whites.

Many of the aborigines living on settlements, missions and cattle stations spend a considerable part of their time away from their designated place of abode. In general it may be said that the longer a settlement has been

established the more time is spent there by its inhabitants, whilst at the other extreme, aborigines living in areas close to the more remote and recently established settlements may spend little of their time at the settlements.

Difficulties.

At the time when these surveys were carried out there was no universally adopted system of naming and identifying full-blooded aborigines. Aboriginal names are of great complexity and subject to various tribal sanctions, and in any case are dependent on the phonetic interpretation of the settlement authority or the examining

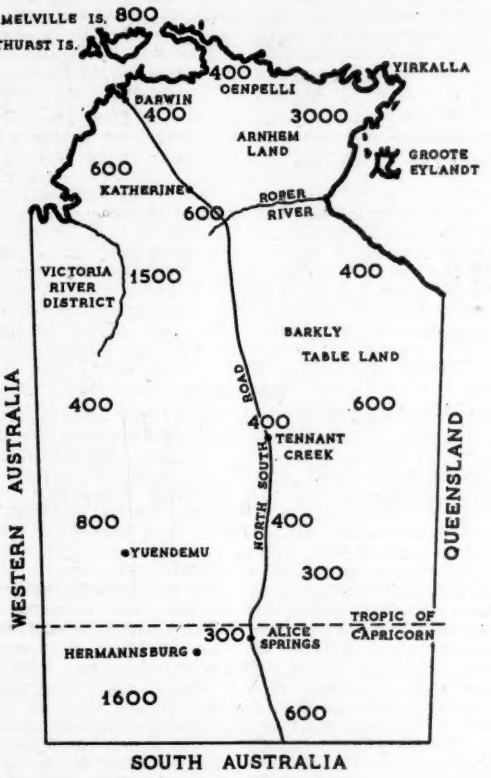

FIGURE I.

This map shows the approximate distribution of full-blooded aborigines in the Northern Territory.

medical officer. Many aborigines are known by a name indiscriminately bestowed at the whim of the nearest white man; consequently names such as Polly, Joe, Banana, Frog, may apply to many different people. With the breaking down of old tribal restrictions on movement the aboriginal today satisfies his nomadic instincts by frequently changing his place of abode, his work (if any), and very often his name. There are still quite a number of aborigines who are afraid of white man's medicine and who could not be examined. It was also found during the course of the Mantoux survey that many aborigines tested failed to report for the reading of the test. In practice many of these were reexamined at a later date, but they have not been included in these published results. Because of these difficulties it has been hard to maintain the accuracy of the card index system, and although it is now considered that the great majority of aborigines have been Mantoux tested, these published figures deal only with aborigines who have been positively identified without any possibility that they were included in the results of earlier or later surveys in any one area. These figures thus deal with the results obtained in 5259 aborigines so tested. These tests were carried out over a period of some three and a half years and in most centres of aboriginal population throughout the length and breadth of the Northern Territory.

Technique.

The technique employed was as follows. An amount of 0·1 millilitre of a one in 1000 dilution of old tuberculin prepared by the Commonwealth Serum Laboratories was injected intradermally into the skin of the forearm. For convenience the test was read in forty-eight hours. A raised area over five millimetres in diameter was taken to indicate a positive result. When possible a subject who failed to react was vaccinated with B.C.G. vaccine.

Results.

Table I shows the over-all results.

TABLE I.

Results of Mantoux Surveys amongst Full-Blooded Aborigines in the Northern Territory.

Age Group. (Years.)	Number Tested.	Positive Results.	Negative Results.
0 to 4 ..	896	148 (16·5%)	748
5 to 9 ..	773	227 (29·4%)	546
10 to 14 ..	566	247 (43·6%)	319
15 to 19 ..	534	292 (54·7%)	242
20 to 24 ..	442	250 (56·6%)	192
25 to 29 ..	496	307 (61·9%)	189
30 to 34 ..	397	255 (64·2%)	142
35 to 39 ..	295	208 (70·5%)	87
40 to 44 ..	219	153 (69·9%)	66
45 to 49 ..	177	121 (68·4%)	56
50 to 54 ..	210	148 (70·5%)	62
55 to 59 ..	145	96 (66·2%)	49
60+	109	76 (69·7%)	33
Total ..	5259	2528	2731

Table II shows the results of similar surveys performed by King (1951) in Western Australia, and Table III by Macken (1952) in Queensland.

TABLE II.

Results of Mantoux Survey in Western Australia.
(After King, 1951.)

Age Group. (Years.)	Positive Results.	Negative Results.	Total.
0 to 5	34 (11%)	256	290
6 to 10	66 (21%)	242	308
11 to 15	101 (41%)	144	245
16 to 20	134 (57%)	100	234
21 to 25	96 (47%)	106	202
26 to 30	152 (61%)	94	246
31 to 35	79 (58%)	56	135
36 to 40	74 (53%)	64	138
41+	455 (53%)	404	859
Total	1191	1466	2657

Tables IV and V show the results obtained in groups of aborigines living in long-established settlements close to white centres of civilization and in recently established settlements remote from prolonged contact with white civilization. In these tables the two settlements from the Central Australian area — Hermannsburg, settled about 1880, and Yuendumu, settled about 1945—have been compared. Two settlements from the Arnhem Land area—Oenpelli, settled in the 1890's, and Yirrkala, settled about 1939—have been compared.

It can be seen that, in general, the rate of conversion is higher and occurs at an earlier age in those aborigines who have had prolonged contact with white civilization and who are living a more or less settled existence.

Table VI shows the results of a Mantoux survey conducted amongst children attending the Darwin schools. In this table the school children have been classified as white and coloured. The term "coloured children" includes all part-aboriginal children, but excludes children of pure Chinese descent. A pronounced difference in conversion rates among these groups can be seen. By comparing relevant age groups in Table I and Table VI it can also be seen that the conversion rate among aboriginal children is very much higher than among white children.

A survey to determine the case incidence of tuberculosis has been commenced and although the results are beyond the scope of this paper it has been found that there is a definite association between numbers of proven cases and the Mantoux conversion rates in any one area.

TABLE III.
Results of Mantoux Surveys amongst Aborigines.
(After Macken, 1952.)

Age Group. (Years.)	Number Tested.	Positive Results.
0 to 1	400	13 (3·25%)
1 to 7	1251	232 (18·1%)
8 to 14	1387	602 (45·78%)
15 to 21	1298	885 (65·35%)
22 to 45	1412	1126 (79·8%)
45+	878	710 (80·8%)
Total ..	6626	3568 (53·8%)

TABLE IV.
Comparison of Findings in Hermannsburg and Yuendumu.

Age Group. (Years.)	Hermannsburg.			Yuendumu.		
	Number Tested.	Negative Results.	Positive Results.	Number Tested.	Negative Results.	Positive Results.
0 to 4	74	51	23	73	68	5
5 to 9	68	28	40	57	51	6
10 to 14	57	10	47	21	17	4
15 to 19	33	2	31	20	10	10
20 to 24	24	5	19	18	10	8
25 to 29	18	2	16	25	13	12
30 to 34	21	2	19	16	6	10
35 to 39	20	2	18	13	4	9
40 to 44	11	—	11	9	4	5
45 to 49	9	—	9	11	5	6
50 to 54	4	—	4	3	3	—
55+ ..	13	—	13	18	6	12
Total	352	102	250 (71%)	284	197	87 (31%)

TABLE V.
Comparison of Findings at Oenpelli and Yirrkala Missions.

Age Group. (Years.)	Oenpelli Mission.			Yirrkala Mission.		
	Number Tested.	Negative Results.	Positive Results.	Number Tested.	Negative Results.	Positive Results.
0 to 4	19	14	5	57	55	2
5 to 9	10	6	4	31	30	1
10 to 14	6	1	5	39	32	7
15 to 19	6	1	5	35	26	9
20 to 24	11	4	7	24	17	7
25 to 29	21	11	10	21	16	5
30 to 34	15	5	10	20	16	4
35 to 39	1	1	—	7	5	2
40 to 44	18	1	17	3	3	—
45 to 49	—	—	—	6	3	3
50 to 54	—	—	—	2	2	—
55+ ..	—	—	—	7	5	2
Total	107	44	63 (59%)	252	210	42 (17%)

TABLE VI.
Results of Mantoux Surveys amongst Children Attending Darwin Schools.

Age Group. (Years.)	Number Tested.	Positive Results.	Negative Results.
All Pupils.			
5 to 9 ..	509	52 (10·2%)	457
10 to 14 ..	383	68 (17·7%)	315
15 to 19 ..	54	16 (29·6%)	38
Total ..	946	136	810
White Children.			
5 to 9 ..	360	20 (5·5%)	340
10 to 14 ..	245	25 (10·2%)	220
15 to 19 ..	28	7 (25·0%)	21
Total ..	633	52	581
Coloured (Part-Aboriginal) Children.			
5 to 9 ..	113	28 (24·8%)	85
10 to 14 ..	91	40 (43·9%)	51
15 to 19 ..	11	6 (54·5%)	5
Total ..	215	74	141

Conclusions.

There appears to be little doubt that tuberculosis was an unknown disease among full-blooded aborigines before white settlement. Holmes (1912) noted the prevalence of cases of tuberculosis among aborigines in contact with white settlements, and then went on to say that he had found no trace of this disease among secluded and uncivilized blacks. King (1951), in a survey in Western Australia, noted little evidence of exposure to tuberculosis among remote groups of aborigines. The results of this Mantoux survey have shown that exposure to tuberculosis occurs at an early age, and more commonly among aboriginal children than among white children and among part-aboriginal children than among white children. The survey has also shown that prolonged contact with white settlements combined with a change from a nomadic to a settled existence has resulted in an increase in the positive Mantoux reaction rate, which occurs at an earlier age. There is little doubt that poor living conditions have done much to assist this spread of tuberculosis. Among whites better living conditions, assisted by vigorous case finding and treatment, have lessened the incidence of tuberculosis; but the disease still remains a serious problem among the aborigines.

Summary.

1. The results of Mantoux tests on full-blooded aborigines in the Northern Territory are presented.

2. The results are compared with the results of similar tests among other groups of people.

3. The results show the influence of white settlement and changed living conditions on the conversion rate among aborigines.

Acknowledgements.

The permission of the Director-General of Health to publish this paper is acknowledged. Thanks are due to Dr. H. Roche for guidance and assistance.

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PROCTALGIA FUGAX.

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THE condition *proctalgia fugax*, consisting of attacks of severe rectal pain, usually nocturnal and occurring without any apparent organic disorder, is of interest to me because I have met with more cases in a short period of practice than is usual. The condition is not well known medically, although it is considered common, and its recognition will save the patient much mental stress, although the attacks may be unrelieved by treatment. It deserves attention because of the dramatic appearance of the patient in the attack, collapsed and looking critically ill, with his complete recovery in a few minutes, and because of the unknown ætiology and the severity of the pain.

Proctalgia fugax is now the accepted name, having been first proposed by Thaysen (1935*a*, *b*). The resistance of the scholars to its mixed Greek-Latin derivation has not lessened its popularity (Ewing, 1953). Thaysen used this name (fleeting rectal pain) because it did not imply any particular ætiology or origin. The first description of the condition is that of Myrtle (1883), but his article does not appear to have been widely read. Curling (1871) has been credited with an earlier report; but the condition he described as rectal neuralgia does not resemble *proctalgia fugax*. MacLennan (1917) wrote of rectal crises of non-tabetic origin; he wrote when *tabes dorsalis* was a common disease and not the rarity it is now. Wallis (1909) and Pybus (1910) were other early writers, but the work of the latter was not published. This was unfortunate because Pybus had a wide interest in and knowledge of the condition, but his article was studied and quoted by Ewing (1953). Thaysen's writings in 1935 and 1936 made the condition much better known, and since then the literature has increased considerably, although it is still thought that many doctors are not familiar with *proctalgia fugax*.

Reports of Cases.

It is proposed to record here a number of illustrative case histories. No local disease process has been found in these cases to account for the attacks of *proctalgia fugax*.

CASE I.—A medical practitioner in his thirties has had attacks of *proctalgia fugax* since the age of eighteen years, occurring twice or three times a year, but already showing some decrease in frequency with increasing age. The bouts, which are always nocturnal, arise irregularly and are brought on by sexual frustration and possibly by nervous tension. The pain is situated two or three inches inside the anus, is sickening like the pain of testicular trauma, and is very severe although he has not felt collapsed with it. The attacks last five minutes or less, with associated priapism at times. He does not suffer with other functional conditions, but is a very restless, intense person. He can sometimes relieve an attack by drawing up the legs, but usually walks about.

CASE II.—This patient is a medical practitioner in his thirties. His attacks began at the age of twenty-five years, and he has had as many as 20 bouts a year, but lately only one or two annually. He also has migraine and thinks that attacks of each condition occur in periods of nervous tension; the reduction in frequency of the bouts is considered due to increased mental contentment following his settling in a good practice after a period of uncertainty concerning his work. He relates his attacks to activity of the pelvic floor muscles, after sexual activity and straining at stool. The bouts are always nocturnal, and last between two minutes and half an hour. The pain is severe, situated about six inches above the anus and boring in nature. He has a hypertonic personality, and is over-conscientious and worrying. There is a family history of asthma. He feels that the genupectoral position relieves attacks.

CASE III.—A clerk, aged forty-eight years, has had attacks of *proctalgia fugax* for thirty years. He has a fear of rectal cancer and presents after most attacks of *proctalgia fugax* for examination and reassurance. He has had as many as six attacks yearly, but the frequency has decreased as he has aged. He considers that bouts are brought on by a full bowel, a cold night, or "intercourse or lack of". They are nearly always nocturnal, and are unrelated to nervous tension. The duration of attacks is several minutes, but on occasions he has not had relief for an hour. The pain is severe, situated three inches or so above the anus, but he has not felt collapsed during an attack. There is no priapism. He has a peptic ulcer, and has had an attack of *alopecia areata*, and although he is fat and apparently jovial, this appearance masks an anxious personality. He relieves an attack by taking food or drink. This usually produces complete relief, although in a severe attack some pain will persist after the gastrocolic reflex has given partial relief. His son also has attacks of *proctalgia fugax*.

CASE IV.—This patient is a salesman, aged forty-two years. His attacks of *proctalgia fugax* began at the age of twenty-two years, and he has 10 bouts or less a year. The pain is a severe dull ache situated "right inside the anus". There is no priapism. He has suffered several other painful conditions, but the pain of *proctalgia fugax* is the most severe in his experience. He also has tension headache, and considers that attacks of headache and *proctalgia fugax* are both likely to occur in periods of nervous tension. He has been treated for an anxiety state and is a very tense, worrying man. His bouts of *proctalgia fugax* have been lessened in frequency by sedation, and the remaining attacks are eased by a drink of milk.

CASE V.—A medical practitioner in his forties has had one to three bouts of *proctalgia fugax* per year for many years. They are irregular, but have not decreased as he has become older. He does not know of any condition which will precipitate attacks, which are usually nocturnal and last from one to five minutes. The pain is severe and "spasmodic", situated two or three inches above the anus, but does not cause faintness or collapse. He is an intense, energetic person, who has both tension headache and true migraine. He has found nothing which will relieve an attack.

CASE VI.—A surveyor, aged fifty-four years, has had as many as 20 attacks of *proctalgia fugax* per year, although he had one period of three and a half years without an attack. He believes that the bouts are brought on by nervous tension, and he is an anxious type of man, having frequent headache and diarrhœa for which no organic cause can be found. He says the pain of proctalgia is "frightful", and that he cries and moans with it. Bouts have been decreased in frequency by sedation, but he knows no way of easing an attack once it has begun. He has had the complaint since the age of twenty-one years, and finds that the bouts usually occur at night, lasting up to half an hour. He was a prisoner of war of the Japanese for three and a half years during the recent war, and did not have an attack while in captivity, although his general health, both physical and mental, became poor. After repatriation the bouts became so frequent that they occurred almost daily, but he did not seek advice until 1952. After investigation, diagnosis and reassurance, the attacks became so rare, slight and fleeting as to be negligible.

CASE VII.—A medical practitioner in his thirties has had attacks of *proctalgia fugax* since his early twenties, related chiefly to sexual activity and constipation, and nearly always occurring at night. The duration of the attack is about fifteen minutes, an occasional attack lasting as long as half an hour. The severity of the pain varies from a mild degree which does not prevent the patient from walking about, to severe boring pain well inside the anus, inducing nausea, vomiting, and collapse, and sometimes loss of consciousness. There is often associated priapism. The attack is eased by the local application of heat, but glyceryl trinitrate (1/200 grain) gives relief more quickly. He is a tense person, subject to tension headache and asthma. His father also has attacks of *proctalgia fugax*.

CASE VIII.—An Army officer in his forties has had attacks of *proctalgia fugax* since the age of thirty-three years, and may have two bouts a week, or there may be a month between attacks, which are quite irregular. The severity also varies; frequent attacks are of mild degree, and the first bout after a longer interval is severe, causing sweating and nausea, although he seems to be more than usually tolerant to pain. There is no apparent precipitating cause and he is a sedate person. Less than five bouts have occurred by day. The pain is situated about two inches inside the anus, and is more severe on the left side. It lasts from five to fifteen minutes, and is always accompanied by painful priapism. During the attack he feels the need to evacuate his rectum but cannot do so, and draws up his knees to ease the pain. Taking food or drink does not give relief, but glyceryl trinitrate (1/200 grain) will ease the pain within a minute or so. He has shown an allergic reaction to penicillin, but otherwise has no allergic or functional condition. X-ray examination with a barium meal and fluoroscopic screening have revealed an hiatus hernia. He

remarks a decreasing frequency of attacks as he ages. He has been very fully investigated for this pain on two occasions, apparently because the practitioners concerned were unfamiliar with *proctalgia fugax*. He gives a concise, exact history on which the diagnosis can be made, to be confirmed by negative results to investigation. His sister also has attacks of *proctalgia fugax*.

CASE IX.—A medical practitioner in his fifties began having attacks of *proctalgia fugax*, almost weekly, in his late forties, but they have now become less frequent, occurring perhaps four or five times a year. He does not know the reason for the onset of the attacks, which almost always occur at night, and have on some occasions been very severe. He has not become unconscious, but has had to lie on the floor in two attacks because he felt he was about to faint. One of these bouts occurred in an office by day, and the office staff became anxious, as they thought that the patient was dying. The bouts last ten minutes or a little more, and the pain seems to involve the whole rectum, which seems to be in spasm. He has not found any method of relieving an attack. He is a restless, energetic and over-conscientious practitioner, but has no other functional or allergic condition.

CASE X.—This patient is a medical practitioner in his sixties. His attacks of *proctalgia fugax* began just before the age of thirty years. His history is peculiar in the frequency and severity of his attacks, which still occur often. He has often had two bouts a week, and in many of these he loses consciousness. The attacks have occurred sufficiently frequently by day to interfere with his professional life, and in this respect his case is similar to that of a famous lawyer (described by M(edico) c P(roctalgia) F(ugax), 1935) who was forced to give up his court work because rising to examine witnesses would induce an attack of *proctalgia fugax*. The features of Case X are otherwise usual. The patient has mild allergic asthma and has had an attack of *alopecia areata*. A son also has attacks of *proctalgia fugax*.

Symptomatology.

As has been indicated in the foregoing case histories, the attacks may occur by day, but are usually nocturnal. They occur at irregular intervals, with sometimes a year or more between succeeding bouts, but commonly several attacks are experienced each year. Usually the onset is without apparent precipitating cause and while the patient is in normal health. Some people state that there are precipitating causes, such as preceding sexual activity (MacEwen, 1935; Blyth, 1935) or a period of nervous tension, fatigue ("Biologist", 1952) or depression. The patient is awakened from sleep by the pain, which is always felt in the same place by one individual, in the rectum, usually four or five inches above the anus. The pain is at first slight, but gradually becomes more severe, reaching a *crescendo* often of extreme severity (Carter, 1935) before slowly passing off. The duration of the attack may be an hour, but is usually less and most commonly five or ten minutes. The pain is variously described, often as a gnawing, agonizing cramp, but the description is constant for each patient, and limited only by the vocabulary of the patient (Murray, 1951). When examined in the attack, the patient may be collapsed, pale and sweating, gasping for breath and may appear gravely ill. He may have lost consciousness. This dramatic appearance, followed in a few minutes by return of colour and recovery, makes this functional condition one of great interest. One patient, a doctor, who had attacks of *proctalgia fugax* and biliary colic, preferred the latter as it was less painful ("Spasm", 1931).

Thaysen (1935*a*) describes an associated sensation occurring in the precordium or epigastrium as a part of the condition, but I consider this to be a result of the severe pain, an accompanying feature to the nausea and collapse. At the peak of the attack there may be painful priapism, but other radiation of pain denotes another condition.

One person will have attacks of varying severity, some being minor or abortive. Frequent bouts will often be less severe, more widely spaced attacks of full vigour. As with migraine, the attacks tend to decrease in severity and frequency with increasing age. There is no residual effect from the attack, except a profound sense of relief and fatigue, and there is no disturbance of intestinal function, either constipation, diarrhœa, tenesmus or paræsthesia in the rectum or anal region.

Incidence.

The condition is far more common than reports in the literature indicate, the number of reported cases being less than 150. It is not mentioned in many standard text-books of medicine and surgery. Thaysen (1935*a*, *b* and *c*, 1936) describes several cases and Pybus (quoted by Ewing, 1953) has seen or heard of numerous sufferers. On the other hand, some doctors write of seeing only one or two cases in a lifetime of medical practice. I have recognized about one case a year for the past ten years, and I consider these not to be diagnoses arising from excessive enthusiasm as the differential diagnosis is relatively simple. The present series includes six of these cases, the records of the remainder not being accessible, and patients referred by colleagues have been included. The sex incidence—the condition is twice as common in males as in females—is not reflected in the present series, as my work is with a predominantly male practice. Ewing (1953) thinks that the condition is very common, but that it does not cause sufficient disturbance to warrant a medical opinion. This would also explain the apparent frequency of doctors among the sufferers, it being believed that they seek advice of their colleagues more readily than do lay patients. Six of Thaysen's ten patients were doctors, as are six of the present series of ten.

Ætiology.

While there has been little text-book reference to *proctalgia fugax*, there is a comparative abundance of opinion about its ætiology. Myrtle (1883) described patients suffering for years from these attacks and "neither they, their medical men, nor consultants even, have been aware of the nature of the mischief". Myrtle had never found any sign of local lesion in these cases, and today this still obtains. Although some cases have been published in which there were local pathological changes, this is considered to be incidental to, rather than associated with, the *proctalgia fugax*. No persisting alteration from normal in the rectum or pelvis has been found in this series, or reported in the literature. Minor differences, probably within normal range, are recorded.

The most attractive proposition is that *proctalgia fugax* is caused by a spasm of the bowel wall at or below the recto-sigmoid junction but above the internal *sphincter ani*, occurring in persons of hypertonic or neurolabile personality, similar to the muscle spasms at various sites to which these people are subject. There is considerable evidence to support this proposition beside the lack of abnormality found on examination. Some writers believe that the spasm is limited to the sphincters of the anus, but this cannot be accepted because of the persistent finding of relaxed anal sphincters during the attack, an observation first made by Nothnagel in 1898, confirmed in the present series of cases. More commonly the site of the spasm is thought to be either in the *levator ani* muscle, or in the musculature of the lower bowel. I favour the latter site, and reasons for this preference will now be advanced. The patient feels the pain to be in the bowel, and while patient's observations are often unreliable, in this case there is considerable evidence to support his area of pain reference.

Smooth muscle is more susceptible to sustained contraction and spasm than skeletal muscle (Best and Taylor, 1940). The relief many sufferers obtain in an attack by initiating the gastro-colic reflex (Pybus, quoted by Ewing, 1953; Bett, 1952; Hughes, 1952) by taking food or a drink is also good evidence that the spasm is in gut—one can imagine the wave of mass peristalsis sweeping down the bowel, forcing the fæcal matter into the rectum and relaxing the spasm. The relief of the spasm obtained by the use of amyl nitrite (Marshall, 1935) or glyceryl trinitrate (Karras and Angelo, 1951) also points to a spasm occurring in smooth muscle. Also it is thought that at the recto-sigmoid junction the anatomical structure is conducive to spasm, there being at this point a definite thickening of the circular muscle fibres of the bowel—the pelvi-rectal sphincter (Best and Taylor, 1940).

The nerve supply of the lower bowel and the *levator ani* muscle also indicates that the pain of *proctalgia fugax* is

a bowel pain (Martin, 1917, 1919, 1920). The innervation of the areas above the pectinate line (this line being the embryological junction of the entodermal part of the anal canal and the ectodermal part, and situated less than one inch from the external opening of the anus) is derived from sympathetic fibres, and below the line from spinal nerves with some sympathetic fibres ("Gray's Anatomy", 1954). With infection, irritation or injury of the portion below the pectinate line, spasm of the sphincter muscles results, with contraction of the anus. A lesion above this line is associated with a relaxed anus, as is found in *proctalgia fugax*.

The principle of contrary or reciprocal innervation, whereby relaxation of the anal sphincters by the pelvic (inhibitory) nerve is accompanied by excitation of the musculature of the bowel wall, suggests that the relaxed sphincter found in attacks of *proctalgia fugax* indicates a spasm of the bowel wall musculature in the rectum or sigmoid colon.

The report by Bolen (1943) of a sigmoidoscopy performed during an attack of *proctalgia fugax* is often quoted as evidence that the spasm is situated in the *levator ani* muscle. Bolen found spasm of the central portion of this muscle, and pressure at this point caused further spasm of the muscle, increasing the patient's pain. His report is not wholly acceptable, because of the difficulty in feeling the *levator ani* muscle (except at its insertion into the sphincters of the anus) through the bowel wall with the sigmoidoscope. Bolen also reported increased redness of the rectal mucosa at the recto-sigmoid junction, and had difficulty in defining the lumen of the bowel, so that he was unable to continue the examination beyond this point. These findings could indicate a bowel wall spasm at the recto-sigmoid junction, although difficulty in locating the lumen of the bowel at this point is not unusual in a normal sigmoidoscopic examination. Bolen's examination, made during an attack of *proctalgia fugax*, was not made under favourable circumstances, and is not considered to be overwhelming evidence for the *levator ani* muscle as the site of the spasm, although it has been much quoted as such.

A relaxed sphincter and a normal rectum on digital examination have been reported many times by medical sufferers, and it is assumed that they cannot reach as high as the site of the spasm. The common occurrence of attacks soon after the pelvic floor muscles have been excited (Sheppard, 1952), as by sexual activity or passage of a constipated motion, suggests that the spasm may be in the *levator ani* muscle, as does the nocturnal preponderance of attacks, which occur in the period of increased susceptibility to cramps of other skeletal muscles, when the patient is warm in bed. Indeed, some patients have likened the spasm of *proctalgia fugax* to the muscle spasm experienced in the forefoot or calf, usually a nocturnal happening. However, the relation of other muscle cramps, bed warmth and coitus to *proctalgia fugax* may be fortuitous, as the swallow and the summer.

The relief obtained by an over-emphasized genu-pectoral position (Thaysen, 1935*a*; Smith, 1935; Bull, 1952) may indicate that stretching the *levator ani* muscles eases the spasm; but more commonly the attack is relieved by the use of the gastro-colic reflex, and the latter mechanism suggests that the spasm is in the musculature of the bowel wall. There are many other theories concerning ætiology in the literature, but none of them are attractive, as they are generally based on a single case.

Psychological Aspects.

At least two of three common factors are present in all cases—the personality type, the coexistence of other functional disorder, and familial tendency. These three factors may all arise from the same basic source. The personality is variously described (Larkin, 1943; Tecon, 1935; Pruitt, 1944), but actually is identical with the migrainous personality, that of the irritable perfectionist. The attacks of *proctalgia fugax* are, as in migraine, less frequent and severe with increasing age, and are more frequent in times of worry, tension, fatigue and boredom. In one case in this series there is a relationship in time between the attacks of migraine and *proctalgia fugax*, both presenting in times of nervous tension. It is also interesting to note that attacks of both conditions are prone to occur in some persons when they feel in good health, mentally and physically. Actually this mental state may vary from a feeling of peace and well-being to an elevation of mood reaching a degree of hypomania, depending on the individual personality; but in any case the subject feels extremely well, and it is while he is in this state that an attack of either *proctalgia fugax* or migraine may occur.

The coexistence of tension disorders and *proctalgia fugax* is striking, particularly that of migraine, asthma, *alopecia areata* and tension headache. Seven of the ten patients in this series have also one of the foregoing conditions. Peptic ulcer, "irritable colon" and eczema occur less frequently. The familial tendency ("C.G.E.", 1931; Thaysen, 1935*a*; Carlill, 1935) is due to the inheritance of a predisposing personality, and four of the ten patients in this series have another member of their family (a parent, child or sibling) with attacks.

Murray (1952) believes that there is little difference, except in the viscus concerned, between *proctalgia fugax*, "gastralgia, enteralgia, colalgia" (as described by the older physicians), *globus hystericus*, spastic colitis, pseudo-angina (Beckman, 1935), many attacks of asthma or dysmenorrhœa, and many other psychosomatic conditions.

Differential Diagnosis.

The most important condition to be excluded is carcinoma of the rectum, not because it is clinically similar, but because it is usually the patient's own diagnosis. Sigmoidoscopic examination will remove this fear. The description of the attack, and the occurrence of bouts over some years, usually make the diagnosis easy; but there are some similar conditions which should be excluded. The most similar is rectal neuralgia (Gant, 1923), usually occurring in constipated persons, and distinguished by a radiation of pain to the hips, perineum and legs (Thaysen, 1936). It is intermittent and remittent and lasts for minutes to days, and the patient may have several attacks in one day (Gant, 1909; Schmidt, 1913). *Proctalgia fugax* lacks this radiating quality, and the bouts are of more constant duration.

Rectal tabetic crises are now rare, but earlier writers suggest the need for serological tests to exclude *tabes dorsalis* (Howard, 1935), as do present-day American writers (together with a battery of other investigations). In tabetic rectal crises the pain lasts longer and the bouts are more frequent. There are often the passage of frequent and watery stools, hæmorrhage and tenesmus, and other evidence of *tabes dorsalis* will be found (Marie, 1904; Lewandowsky, 1911).

With coccydynia the patient is most uncomfortable while sitting, and has a dull ache at the base of the spine which is eased by moving. It is more common in females, unlike *proctalgia fugax*. Ball-valve accumulations of hard fæces are easily excluded. The causes of anal pain—fissure or ulcer, prolapsed internal pile, perianal hæmatoma or abscess, and tenesmus resulting from dysentery—are excluded by the anal spasm associated with them.

Treatment.

Treatment of the attack consists of relieving the muscle spasm, and several methods may give relief. The two methods most commonly used are the local application of heat and the use of the gastro-colic reflex, the latter initiated by the taking of food or a drink (Pybus, quoted by Ewing, 1953; Bett, 1952; Hughes, 1952). In many cases Ewing (1953) reports that the use of the gastro-colic reflex works "as if by magic". It has not been as successful in my experience, although if it is not immediately effective, it does ease the pain to some extent. The local application of heat is often used (M(edico) c P(roctalgia) F(ugax), 1935; "Neuralgia", 1931), and the most effective method is a very hot bath. Precautions should be taken against collapse in the bath. Some people obtain relief by adopting certain positions, usually an exaggerated genu-pectoral position (Thaysen, 1935*a*; Smith, 1935).

Another cure with its adherents is the injection of air or water into the rectum, and a syringe may be carried about on the person for this purpose (Marshall, 1935; Ryle, 1948). The inhalation of amyl nitrite has been reported as effective (Marshall, 1935), and glyceryl trinitrate tablets, 1/100 or 1/200 grain, are used by Granet (1946) with good results; this was confirmed in the series of 12 cases published by Karras and Angelo (1951), as six of their patients who used glyceryl trinitrate obtained dramatic relief of the pain, usually within a minute, while another patient considered his attacks to have been lessened in severity. An attempt at defæcation, which is usually unsuccessful, may ease the pain (Carlill, 1935), also micturition (if possible, sometimes it is not) is reported to relieve an attack (Hurst, 1943).

General measures of reassurance that no serious disease exists, and sedation if the bouts are frequent, will assist. Reassurance in one case in this series almost abolished the attacks. The exhibition of morphine may be necessary in severe cases (Abrahams, 1935), and chloroform was used by MacLennan earlier in this century. If a precipitating cause is found, it is dealt with on its merits (Weber, 1943). There are several other suggested remedies (Tecon, 1935; Yeomans, 1936), but they appear to apply only to the isolated case.

Summary.

The incidence of *proctalgia fugax* is discussed, and a description of the attack is given. The cause of the pain is considered to be an excessive and sustained contraction of the bowel wall musculature at a point at or below the recto-sigmoid junction, but above the internal sphincter of the anus, occurring in persons of hypertonic personality. The differential diagnosis is simple, and if the condition is known, it should not be missed. Relief of the attack may be obtained by the local use of heat, by the administration of glyceryl trinitrate, or by initiating the gastro-colic reflex by taking food or a drink. The condition is sufficiently common to be included in the differential diagnosis in cases of rectal or ano-rectal pain. Illustrative case histories are given.

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Reports of Cases.

A CASE OF CONGENITAL NON-SPHEROCYTIC HÆMOLYTIC ANÆMIA PRESENTING WITH AN APLASTIC CRISIS.

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THERE have recently been described several cases of congenital non-spherocytic hæmolytic anæmia. Dacie (1954) has reviewed a total of 16 families suffering from this type of anæmia. It differs from the classical hereditary sphero-

cytosis in several important respects. There is no spherocytosis, and fragility tests give normal results. Splenectomy fails to effect a clinical cure. Pappenheimer bodies and target cells may be a feature of the blood film after splenectomy. Associated with the hæmolytic process there is a reticulocytosis, which in one case described by Dacie constantly exceeded 60%. Jaundice at birth has been described.

Clinical Record.

A., a boy, aged six years, was admitted to the Cairns Base Hospital on November 9, 1954, with pronounced pallor and listlessness. The hæmoglobin value was 4·0 grammes *per centum* on the previous day.

The child had been jaundiced at birth, and was given a blood transfusion at the age of ten weeks. At the age of seven months a diagnosis of hereditary spherocytosis was made and splenectomy was carried out. The child had remained in apparently good health until his present admission to hospital, but no hæmatological investigations were made.

The family is descended from British stock, and did not come from the Mediterranean littoral. The eldest sibling, a boy, was yellow at birth, and died at the age of three years with anæmia. A. is the second child. The third child is a girl, aged five years, and has shown no signs of anæmia; the hæmoglobin value, the number of reticulocytes, and the results of fragility tests were all normal, as was a blood film examination. The younger sister developed severe anæmia one month after birth; investigations at this time showed a hæmoglobin value of five grammes *per centum*, with numerous reticulocytes. The result of the fragility test was normal. She was maintained on blood transfusions until she was a year old, when she underwent a splenectomy. Shortly after this operation she developed gastroenteritis and died.

On his admission to hospital, the boy had a hæmoglobin value of 3·3 grammes *per centum* and a red cell count of 1,100,000 per cubic millimetre. The hæmatocrit reading was 11 cubic centimetres, making a mean corpuscular hæmoglobin concentration of 30·0% and a mean corpuscular volume of 100 cubic μ. The proportion of reticulocytes was less than 0·1%. The white cells numbered 8000 per cubic millimetre, 51% being neutrophile cells, 38% lymphocytes, 9% monocytes and 2% basophile cells. Examination of a blood film revealed some hypochromia and target cells, and many other red cells had pale crescents without the complete target formation. The plasma protein content

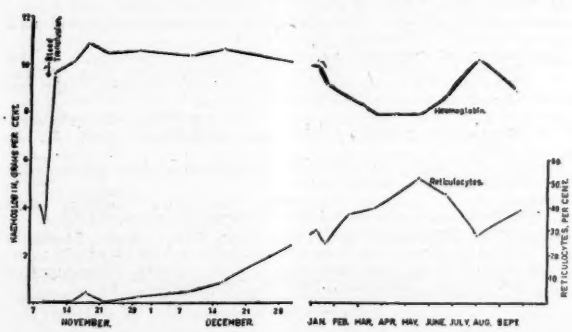

FIGURE I.
Hæmoglobin and reticulocyte levels of patient A.

was 6·5 grammes *per centum*. A sternal marrow examination revealed an almost complete absence of primitive red cells, apart from proerythroblasts which were slightly increased in numbers (2·5% of nucleated cells). There was no aplasia of the white cells, and the number of plasma cells was slightly increased. Two pints of compatible blood were given, and the hæmoglobin value was 10·1 grammes *per centum* on November 15. The hæmoglobin value remained at about this level for a month and then slowly started to fall, reaching a level of 7·9 grammes *per centum* on March 21, 1955; it has remained near this level since (Figure I). Over the same period the number of reticulocytes slowly increased to a level between 40% and 50% (Figure I). Pappenheimer bodies became very numerous, 47% of the cells containing them on March 21. The boy remains well. His mother says that he is always "full of energy", as he had been between the time of splenectomy and the onset of the anæmic crisis.

Osmotic fragility tests, carried out by Creed's method (Whitby and Britton, 1953) and Dacie's method (1950) with buffered saline solutions, both revealed a decreased fragility (Figure II). The fragility after twenty-four

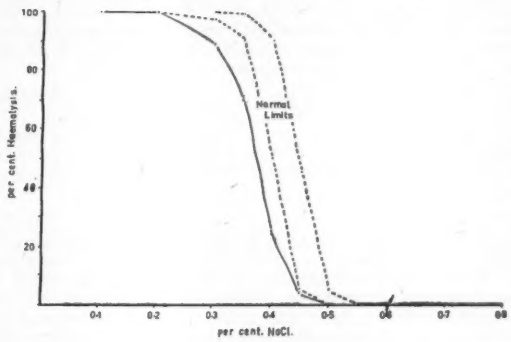

FIGURE II.
Result of osmotic fragility test on patient A. (Normal limits indicated by interrupted lines.)

hours' incubation was within the normal limits (Figure III). Autohæmolysis at twenty-four hours and forty-eight hours was increased about four times the normal (test 4·4% and 9·7%, control 1·2% and 2·2%). On the addition

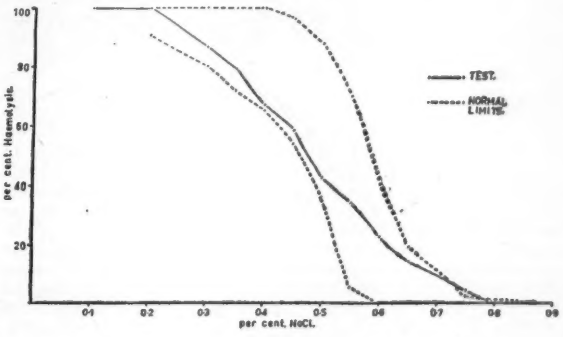

FIGURE III.
Result of osmotic fragility test after incubation of blood for twenty-four hours on patient A. (Normal limits indicated by interrupted lines.)

of glucose this was reduced in the same proportion as a normal control (test 1·9%, control 0·4%). A direct Coombs test produced a negative result. Mr. O'Hagan, of the Queensland Red Cross Blood Transfusion Service, examined the blood for fœtal hæmoglobin, but none was detected either by electrophoresis or by alkali denaturation tests. The bilirubin content on March 21 was 0·8 milligramme per 100 millilitres. Tests for sickling gave negative results.

Further investigation of the family revealed little. Both parents had normal hæmoglobin values, normal responses to fragility tests and normal reticulocyte counts. Ten

close relatives had normal hæmoglobin and reticulocyte values, except for the maternal grandmother, who had a hæmoglobin value of 12·8 grammes *per centum* and a reticulocytosis of 3·1%.

Discussion.

There can be little doubt that this is a case of congenital non-spherocytic hæmolytic anæmia. Dacie (1954) has described two types of this disease. In the second type there is a pronounced increase in fragility of the red cells after incubation. Autohæmolysis is increased 10 to 20 times the normal, and the addition of glucose has no effect on this rate. In the first type osmotic fragility is increased only by the normal amount on incubation. Autohæmolysis is normal, and glucose reduces this, but not by the normal amount. It is not possible to fit this case into either category, though the finding of normal fragility after twenty-four hours' incubation, and an increase in autohæmolysis by only four times the normal value, make it more like the first than the second type.

In the original blood film examined target cells were a pronounced feature, and this suggested the possible diagnosis of Cooley's anæmia, a few cases of this disease having been reported in subjects not of Mediterranean stock. Further investigation, including examination for fœtal hæmoglobin, did not support this diagnosis.

The original diagnosis of hereditary spherocytosis was supported by the child's clinical improvement following splenectomy. However, hæmatological investigations were not carried out, and it is now evident that the child has learnt to live in apparent health with a hæmoglobin value of eight grammes *per centum* and a reticulocytosis of 40% to 50%. It is probable that these blood findings were present before the anæmic crisis. No increased fragility has been demonstrated in the patient's red cells or in those of the family.

The severe anæmia associated with aplasia in the marrow of all red cell precursors except proerythroblasts, and with very scanty reticulocytes, confirms that this child had an aplastic crisis similar to those described in hereditary spherocytosis by Owren (1948) and by Dameshek and Bloom (1948). Owren described six cases in which the marrow was aplastic during the hæmolytic crisis of hereditary spherocytosis, and he suggested that this aplasia, not increased hæmolysis, was the cause of the rapidly developing anæmia. He suggested that the crisis should be called aplastic and not hæmolytic. Dameshek and Bloom (1948) confirmed Owren's findings; but they suggested that a hyperactive spleen acting on the marrow caused the aplasia, and they also considered that there was an exacerbation of the hæmolytic process. Singer, Motulsky and Wile (1950) described two aplastic crises in cases of sickle cell anæmia. After recovery in one of their cases, they were able to show by transfusion experiments that the rate of red cell destruction was sufficient to explain the rapid anæmia that developed during the crisis. In this case no exacerbation of the hæmolytic process was necessary to explain the crisis. Hypersplenism is not a feature of sickle cell anæmia, and these findings support Owren's concept and not the theory of Dameshek and Bloom.

Crises in hereditary spherocytosis have frequently been described as occurring stimultaneously or in rapid succession in several members of the same family. Often there is a history of an upper respiratory infection, and the cause of the crisis is usually considered to be infective. One of Singer and Motulsky's patients with an aplastic crisis in sickle cell anæmia had atypical pneumonia, the other an upper respiratory infection. In the case described here the patient had had a "cold" a week before, but on his admission to hospital there were no signs of respiratory infection.

In most descriptions pancytopenia is described with anæmia, leucopenia and thrombocytopenia, though the examination of the marrow reveals a normal number of primitive leucocytes. Owren in one of his cases found a "shift to the left" in the myeloid cells of the marrow, and considered that this would explain the leucopenia. In the case that I have described there was no leucopenia, and the number of platelets was never estimated. Other cases also have been described in which leucopenia was absent (Emery and Lemmon, 1954). It is possible that the leucopenia observed is the result of hypersplenism, absent in our case; but it is more probable that it is associated with the infective process causing the erythrocytic aplasia.

Gasser (1950) has made an interesting series of observations on the occurrence of aplastic marrow in children not suffering from anæmia. They were suffering from a variety of illnesses including atypical pneumonia, and toxic reactions due to drugs and insect bites. Most of his patients showed allergic signs with pronounced eosinophilia almost suggesting eosinophilic leuchæmia. The eosinophilia is not a feature of aplastic crisis in hæmolytic anæmia. However, the findings Gasser has described suggest that aplasia of the bone marrow lasting for a few days may occur in many types of illnesses. An anæmic crisis will occur only if blood is being destroyed at a rapid rate. In the non-anæmic individual whose red cells have an average life of 120 days, an aplasia lasting for ten days will lower the hæmoglobin value only about one gramme *per centum*. However, in hereditary spherocytosis red cells may have a life of only fourteen days, and similar aplasia in this case will cause very profound anæmia. This theory suggests that an aplastic crisis would occur in any condition in which red cells have a short life. As was mentioned above, crises have been described in sickle cell anæmia, and that they have not previously been described in non-spherocytic hæmolytic anæmia must be due only to the great rarity of this condition.

It is interesting to note in this case that a crisis occurred after removal of the spleen; this is against Dameshek and Bloom's theory that hypersplenism is the cause of the crisis.

Summary.

A case of congenital non-spherocytic hæmolytic anæmia presenting in an aplastic crisis is described. The patient had undergone a splenectomy five years previously.

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Reviews.

Synthetic Drugs: A Handbook for Chemists, Physicians and Pharmacists, by H. Ronald Fleck, M.Sc., F.R.I.C., F.R.M.S.; 1955. London: Cleaver-Hume Press, Limited. 10" × 6", pp. 388. Price: 70s.

"SYNTHETIC DRUGS", by H. Ronald Fleck, which is described as a handbook for chemists, physicians and pharmacists, carries some sixteen chapters, each devoted to a specific therapeutic group of drugs. They range from amœbicides and anæsthetics through antihistamines, antibiotics and hormones to the sulphonamides and vitamins.

These offer a pleasant series of tasty snacks for the scientific dilettante, but the project of supplying a substantial and satisfying meal of synthetic, clinical and analytical data having equal palatability to physician, pharmacist and chemist is, perhaps, a vaunting ambition.

The physician will find the clinical data to be meagre, incomplete, rarely practically informative, and far from up to date. There are, in addition, so many gaps in the normal sequence of therapeutic thought and application that the value of the book as a clinical reference is negligible. As an example, the chapter on "Cardiac Drugs" does not include the cardiotonic glycosides of digitalis, strophanthus and other plants as these are, for the most part, natural products. On the other hand it excludes the many synthetic vasodilators in current use, amyl nitrite being the sole representative. The place of the basic cardiac drugs is taken by a miscellany of "cardiovascular agents" which include such diverse substances as khellin, "Pronestyl", some of the analeptics, some anticoagulants of the coumarol type and some mercurial diuretics.

The pharmacist will find much of interest and much useful chemical reference, but very few practical pharmaceutical data—the various forms in which many of the drugs may be therapeutically presented having little place. There is an aura of inconsistency in the selection of names for the drugs, for, while many are cited under their official or their approved names, some which have official status will appear under proprietary names.

The book is excellently printed, the indices, general and clinical, are adequate, and the various stages in the synthetic development of each drug are set out with a clarity and a graphic presentation that greatly facilitate an appreciation of its clinical constitution and relationships.

This probably will be found to be the most useful aspect of the volume and to justify its place as a work of occasional reference.

Gehirn und Auge: Grundriss der Ophthalmo-Neurologie, by Robert Bing and Roland Brückner; Third Edition; 1954. Basle: Benno Schwabe and Company. 9½" × 7", pp. 248, with 147 illustrations. Price: 28 francs (Swiss).

IN the opening sentence of the third edition of their book "*Gehirn und Auge*" (Brain and Eye), Bing and Brückner point out that the fibres of the optic nerves comprise 38% of the total number of nerve-fibres in the brain—a finding not unexpected in view of the enormous importance of his acute vision to man. The acute, binocular stereoscopic vision which he enjoys is denied to the mammals with the exception of a few primates. This superiority in the faculty of visual perception is accompanied by a very highly developed and complex cerebral cortex. It is not unreasonable to assume that the two are associated and to seek for evidence to demonstrate that the laterally directed eyes of the mammal with a complete decussation of the optic nerves have gradually swung around to the position in man where the eyes look straight ahead and there is a hemidecussation of the optic nerves. At the present time, comparative anatomy can offer nothing to elucidate this problem. The frequent harking back of Bing and Brückner to the anatomy of the lower vertebrates gives a *fin de siècle* atmosphere to their work which is one of its charms and serves as a reminder of those mid-Victorian years when not to follow Darwin was heresy indeed.

A preliminary discussion of the anatomy and physiology of vision leads on to the pathology of the oculo-motor system, of the fifth and seventh nerves and of the visual pathway. The book concludes with a description of diseases of the brain which may affect the eye and this is followed by a detailed description of methods of examination.

In a comparatively small book a neurologist and an ophthalmologist have cooperated in an attempt to encompass the field of neuro-ophthalmology. They have succeeded in supplying an accurate and ready source of reference to the ophthalmologist seeking help in a neurological problem.

The Lacrimal System, by Everett R. Veirs, M.D.; 1955. London and New York: Grune and Stratton. 10" × 6½", pp. 168, with 68 illustrations. Price: $7.50.

THIS monograph deals in a comprehensive manner with all aspects of the lachrymal system. The physiology, both normal and pathological, of tear secretion is considered in detail and the various factors responsible for the drainage of tears down the nasolachrymal duct are stressed. The genetic and developmental anatomy is efficiently dealt with by J. V. Cassady and the importance of this in understanding the anomalies of the system is emphasized.

In regard to disorders of the canaliculus the various operations for repairing the injured canaliculus and the obstructed canaliculus are clearly described, and the functional results of each are criticized. The value of the Kuhnt-Szmanowski operation for ectropion of the lower lid is rightly stressed and the "three snip" operation is emphasized.

Chapters follow on congenital atresia of the nasolachrymal duct, and on the ætiology, clinical manifestation, diagnosis and treatment of dacryocystitis. The technique of probing the nasolachrymal duct both from the canaliculus and from the nose is described together with the author's method of the use of the plastic tubing, introduced through a thin-walled needle, in cases of stenosis of the duct.

Dacryocystorhinostomy is considered in detail, and the author states it results in "cures in practically all instances"—surely an optimistic statement. The monograph concludes with chapters on tumours of the lachrymal sac and lachrymal gland. The author urges the need for radical surgery in the presence of the malignant lachrymal gland tumour.

The book is well written and well illustrated, and presents a balanced and practical account of modern ideas on diseases of the lachrymal system.

Ciba Foundation Symposium on Porphyrin Biosynthesis and Metabolism, edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Elaine C. P. Millar, A.H.W.C., A.R.I.C.; 1955. London: J. and A. Churchill, Limited. 8" × 5½", pp. 320, with illustrations. Price: 30s.

THE latest volume of the Ciba Foundation Symposia contains papers and discussions given at a symposium on porphyrin biosynthesis and metabolism. Twenty-six experts on various aspects of the subject from Europe and North America, including two from Sydney now working in England, met and a series of papers were read and discussed. There are twenty papers which can be divided into three groups. The first group deals with the early stages in the in-vivo synthesis of porphyrins, the second group deals with the fixation of iron to the porphyrins, and the third with the effect of certain drugs on porphyrin metabolism. The subject matter of all the papers is highly technical and could be read with advantage only by those having a very good knowledge of porphyrin chemistry. The discussions on the papers are of a high standard and very helpful. The book gives a splendid account of the present position of porphyrin biochemistry, but would not be very useful to the average physician.

Die Überfunktion der Nebennieren, by Rudolf Chwalla; 1955. Vienna and Bonn: Verlag für Medizinische Wissenschaften. 9½" × 6½", pp. 342. Price: DM48.

THIS is a peculiar book. Its arguments rest entirely on the assumption that an increase in the amount of adrenal tissue, be it in the form of large or small, solitary or multiple, adenomata, or of bilateral or unilateral hyperplasia, is equivalent to a "hyperfunction" of the adrenals. Even certain colour changes of normal sized adrenals are regarded as indicating adrenal hyperfunction. Morphology has, as the author suggests, stolen a march on biochemistry, which has not developed its methods sufficiently far as to be able to confirm these observations by chemical examinations.

To prove his points the author has employed two methods. First, he approaches his material (a very large number of autopsies) from the point of view of what concomitant pathological lesions are present in an above average proportion when an anatomical hyperadrenal state is found. Secondly, the approach is reversed. The author starts with any of a very large number of pathological conditions and then shows in which the adrenal lesions, which according to him signify adrenal hyperfunction, are found more often than in the average population. Amongst these conditions there are not only those that might have been expected (as Cushing's syndrome, certain forms of diabetes and the adreno-genital syndrome) but at least forty others with an extraordinarily wide range.

Out of this immense mass of details and figures emerges then, in the final chapters, the picture of the hyperadrenal individual; a man of pycnic, athletic or "apoplectic" build, with strong bones and muscles, inclined to be obese, his skin rich in abnormal

pigmentation and hair (except for a bald head), a deep thorax and a prominent abdomen. He does not suffer much from exogenous diseases; if he contracts them, he easily throws them off, but he is always threatened by the one weak system in his powerful body—his vascular system. Atherosclerosis in all its varied forms and in all possible organs is his main disease and the one to which he will finally succumb. (Emphysema and hypertrophy of the prostate are the next most common diseases.) All this is, according to the author, genetically determined and characteristic of the individual's constitution. It is therefore not astonishing that the final chapter on therapy is quite short and offers really nothing but the hope that on the basis of this new knowledge therapeutic methods may develop.

An extraordinary book indeed. An enormous amount of work has gone into it, an immense amount of detail has been collected, which is certainly interesting and may be significant, but it does not seem likely that many readers will follow the author's arguments and accept his deductions and conclusions. A very comprehensive index omits, unfortunately too often, the initials of the authors, the titles of the papers and the page numbers of the journals.

The Physiological Basis of Medical Practice: A Text in Applied Physiology, by Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (London), F.R.S., F.R.C.P. (Canada), and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada), F.R.C.S. (Edin.), F.R.C.P. (Canada), M.R.C.S. (England), L.R.C.P. (London); Sixth Edition; 1955. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 1370, with three plates in colour and 80 text figures. Price: £6 9s.

IN the sixth edition of Best and Taylor's "Physiological Basis of Medical Practice" much of the text has been revised, and by means of judicious deletion the size of the book has been reduced. The index has been recast in an attractive manner and is now easier to use. Much of the prestige achieved by this book in the past has been due to the presentation of a nicely balanced selection of information related to all aspects of physiology, and there is little doubt that a similar conclusion is applicable to the new edition.

Operative Technic in General Surgery, edited by Warren H. Cole, M.D., F.A.C.S.; Second Edition; 1955. New York: Appleton-Century-Crofts, Incorporated. 9½" × 6½", pp. 997, with many illustrations. Price: $20.00.

THE first volume of the second edition of Warren H. Cole's "Operative Technic in General Surgery" embraces the abdomen, breast, thyroid gland and some miscellaneous subjects. With a different contributor writing each chapter, the book may be regarded with some exceptions as representing the operative surgery of the American Middle West.

It might well be argued whether such detailed dissection does not defeat its object. It results, for example, in portions of the same operation being described by two writers, as in resection for ulcerative colitis, where the making of the ileostomy is placed in a different chapter from resection of the colon and rectum; and in conflict of advice, as where the writer on abdominal incisions disapproves of the methods advocated by the majority of contributors.

After preliminary chapters on wound healing, shock and hæmorrhage, a very good chapter on burns is contributed by C. C. Lund, of Boston, in which he gives somewhat restrained approval to the popular open methods of treatment.

Amputations are described adequately by McKeever, of Los Angeles, and an especially good account is given of the rehabilitation of the amputee. Professor Coller, of Ann Arbor, contributes a learned article on abdominal incisions, with firm advocacy of the virtues of the transverse incision over the vertical. His meticulous methods of making and closing incisions are, however, rarely followed by other contributors.

A condensed but valuable chapter on the surgery of the stomach and duodenum is written by Walters, of the Mayo Clinic. In marked contrast to most of his co-authors, he discusses the indications and results of alternative methods, and from his immense experience states some surprising facts. In chronic gastric ulcer, though partial gastrectomy is usually indicated, he is opposed to very high resections, since local excision of the ulcer, combined with gastro-jejunostomy, gives very good results. Though this is a purely empirical procedure, the rationale of which is difficult to understand, it may be recalled that some years ago the great English authority, A. J. Walton, in his review of 6000 gastric operations, reached a similar conclusion. In chronic duodenal ulcer also, though partial gastrectomy is to be advocated, when its performance seems perfectly safe, resection of more than half the stomach carries an increasing morbidity which more than counterbalances any reduction in subsequent stomal ulceration that might be achieved by higher resection.

Dragstedt, of Chicago, describes with great clarity his radical subdiaphragmatic vagotomy.

The discussion of acute small gut obstruction, by an associate of Wangensteen, elicits the important finding that after a long experience of non-operative management, early operation is now recognized as carrying less risk and a shorter hospital stay. This chapter also describes fully the so-called aseptic methods of intestinal anastomosis. Coming, however, to the surgery of the colon and rectum, the distinguished authors of these chapters, Roscoe Graham, of Toronto, and Claude Dixon, of the Mayo Clinic, distrust profoundly these methods, as failing to recognize that leakage of an anastomosis is related to defective blood supply rather than infection, and advocate strongly open anastomosis of the ends of gut after wide cutting back on their anti-mesenteric borders.

Warren Cole contributes an adequate chapter on the biliary tract, in which full attention is paid to those anatomical anomalies which may lead to surgical disaster. In his discussion on the surgery of the pancreas, Brunschwig, of New York, describes a method of pancreatic resection in which the stump is occluded by ligature instead of being implanted into the jejunum. Such a procedure might rob this formidable operation of one of its major risks of leakage.

In a good chapter on hernia, by Lehman and Davis, of the University of Virginia, no mention is made of the method of repair of femoral hernia described by Lotheisen fifty years ago.

The surgery of the breast, by Orr of Kansas, includes consideration of the superradical operations by Wangensteen and Urban, in which an attempt is made to extirpate the anterior mediastinal lymph channels.

Authors of great experience in their fields, Pemberton, Sumner Koch and Glenn, describe the surgery of the thyroid gland, the hand and the skin and subcutaneous tissues.

Scant regard is paid here to the place of radiotherapy as an alternative to surgical excision in the common malignant lesions of the skin.

To sum up, this is a good book, describing faithfully the surgery of a geographical area whose practitioners usually place effectiveness above artistry of execution. It contains a number of typographical errors, and to the extent that the results of operative procedures are rarely presented, will probably be of more value to the experienced surgeon than to his juniors.

Introduction to Hepatic Surgery, by Henry Gans, M.D., with an introduction by A. Brunschwig, M.D.; 1955. Amsterdam, Houston, London, New York: Elsevier Publishing Company. London: Distributors, Cleaver-Hume Press, Limited. 9" × 6", pp. 284, with 120 illustrations. Price: 70s.

SINCE George Pack showed his films on partial hepatectomy for cancer in this country, this new adventure upon the frontiers of surgery has commanded much interest. Gans's book supplements the demonstration and brings the important basic sciences into the picture. It is therefore indispensable to those who will embark upon this esoteric surgery.

The indications for hepatic resection are not common in Australia (namely hepatoma, cholangioma, isolated metastatic neoplasms, some gall-bladder cancers, certain injuries and benign newgrowths).

The embryology introduces the concept of the liver as a paired organ—the approach of the surgeon ligating the hilar pedicle, and not in conformity with the external anatomical features. The hilar abnormalities are described in detail, because failure to recognize vital venous connexions may lead to hepatic necrosis.

The surgical technique is clearly enunciated and the pre-operative preparation and after-care are defined. The magnitude of the operation is impressive and the forestalling of complications and the recognition of post-operative mishaps are imperative. Different approaches are recommended for the right (abdomino-thoracic) and left (sternum-splitting mid-line) hepatic resections. It is obvious therefore that the surgeon must know fairly accurately what he expects to find.

This surgical virtuosity is rendered possible by the regenerative capacity of the liver (up to 80%), the proper preparation of the patient (avoidance of hypoproteinæmia, cachexia and hypovitaminosis), the prompt attack on a substantial list of postoperative complications which may occur, and lastly the gentle manipulations and impeccable operative technique.

Written like an essay, this work is authoritative, informative and clearly illustrated. It is a notable contribution to the advance of excisional surgery.

Minor Surgery, by John E. Sutton, M.D., F.A.C.S.; 1955. New York: Landsberger Medical Books, Incorporated. Distributed by The Blakiston Division of the McGraw-Hill Book Company. 8″ × 5¼″, pp. 334, with 102 illustrations.

THIS is one of the books in the series "Hand-books for the General Practitioner" published by Landsberger Medical Books, Incorporated. In it is discussed the treatment of those surgical problems that the author considers may be undertaken safely in the surgery or the patient's home. In describing each procedure, whether it be opening an abscess, setting a fracture, or performing a simple skin graft, the author points out the pitfalls to be avoided as well as giving full practical details of technique. He advocates mainly the use of local anæsthesia, and a useful practical chapter is devoted to this topic. Sets of instruments required are detailed and instructions are given on the various methods of bandaging, and a series of excellent photographs and many line sketches lend added clarity to the text.

The chapter headed "Antimicrobials" runs into eighteen pages and discusses the uses of the various modern antibiotics and includes a most useful table showing the relative sensitivity of common organisms to the different drugs. Minor surgery of infants and children is well described with many useful pointers. While general practitioners who have been in practice for some time will have devised their own particular methods for carrying out most of the minor procedures described, all young doctors can learn much and save themselves many headaches by studying this useful volume.

Rheumatism: Fibrositis, Arthritis, Lumbago, Sciatica, "Slipped Disc", Gout, Spondylitis, by W. S. C. Copeman, O.B.E., M.D., F.R.C.P., and R. M. Mason, D.M., M.R.C.P.; 1954. London: Gerald Duckworth and Company, Limited. 7½″ × 5″, pp. 152, with nine illustrations. Price: 8s. 6d.

THIS small book is one of the "Modern Health" series. The book is written for patients suffering from fibrositis, arthritis, lumbago, sciatica, "slipped disc", gout and spondylitis. It is designed to give these patients reliable information concerning their disease, as regards its cause, symptoms and management. It does not provide information for self-treatment, but allows the patients under treatment for these conditions to have an intelligent understanding of their condition, which is so desirable. Sufferers who read this book will then have no need to search for information in unreliable sources.

The book describes in simple language the conditions mentioned and gives a certain amount of historical information about each. There is an adequate discussion of both the classical home remedies, with information about the latest treatments, including cortisone and ACTH.

A special chapter is devoted to various gadgets which help the crippled person, especially for routine housework.

The authors, acknowledged authorities in this field, are to be congratulated on producing a book which is short and readily understandable by the lay person. It should provide sufferers from these diseases with a more intelligent and healthy outlook on their diseases.

Office Procedures, by Paul Williamson, M.D.; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 11″ × 8″, pp. 422, with many illustrations. Price: £6 5s.

THIS book is written in a friendly colloquial style and gives the author's own ideas on all aspects of many procedures in general practice that can be carried out in the surgery. The book is essentially most practical throughout, and as chapters are devoted to such topics as X-ray technique, physiotherapy and minor pathological investigations and in addition the various bodily systems are covered, it can readily be seen that the scope of the work is very wide. In some ways the scope is perhaps too much to be attempted in one volume, and certainly some of the subject matter is very brief and sketchy. Thus the seven pages devoted to obstetrics are mainly concerned with the diagnosis of pregnancy and the procedures at surgery visits, and this chapter contains nothing more than a final year student should know.

The author describes his own simply improvised gadgets, such as an eyelid retractor made from a paper clip and a simple incubator for bacteriology made from a cardboard box with heating supplied from a light globe, but while these are of interest they are not of much real clinical importance.

In writing on ganglions, the author rather surprisingly still advocates "grandmother's method" and states "this book is about the right size for ganglion smashing"; but he warns that it is possible to fracture the navicular using this "smashing" method. However, if this should happen to the book-wielding surgeon, the work also gives details for X-raying the navicular and also for correctly applying a plaster of Paris cast to immobilize the fracture. Readers will gather that Dr. Williamson must have had a staff of at least three helpers, unless he himself performed all the different chores required to keep the activities listed functioning in his office. Certainly some of the procedures described would serve temporarily in outlying practices where every doctor has to be ingenious at devising impromptu aids, and speaking generally many of the ideas and suggestions are not altogether original, although they are the author's way of doing things. These methods will be a guide for young general practitioners, and despite many gaps the book is sincerely written in a most readily readable manner.

Medical Terms: Their Origin and Construction, by Ffrangcon Roberts, M.A., M.D., F.F.R.; Second Edition; 1956. London: William Heinemann (Medical Books), Limited. 7½″ × 5″, pp. 96. Price: 6s.

IT is barely two years since we reviewed the first edition of this book, and already a second edition has been required. It will be welcomed by those who are interested in understanding medical terms and their origin, in contradistinction to the definition of their meaning. This little book makes delightful reading and is packed full of scholarly information about the terms which are used by medical men every day.

Notes on Books, Current Journals and New Appliances.

Advances in Pediatrics, edited by S. Z. Levine, associate editors John A. Anderson, Margaret Dann, A. Ashley Weech, Myron E. Wegman and Warren E. Wheeler; 1956. London: The Year Book Publishers, Incorporated. Volume III. 9″ × 6″, pp. 273, with illustrations. Price: $8.00.

SEVEN comprehensive and authoritative articles make up this volume. Their subjects are the ætiology of infantile diarrhœa, isosexual precocity in boys, including a case of gonadotropin-producing teratoma, sarcoidosis in childhood, the offspring of diabetic and prediabetic mothers, subdural lesions in childhood with special reference to infectious processes, the prevention of accidents in childhood, and mental deficiency. Subject and author indexes facilitate reference to this useful book.

Clinical Neurosurgery: Proceedings of the Congress of Neurological Surgeons, New Orleans, La; Editor-in-Chief, Raymond K. Thompson, M.D.; 1955. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9″ × 6¼″, pp. 210, with 89 illustrations. Price: 86s.

THIS volume is in fact the proceedings of the Congress of Neurological Surgeons held in New York in 1954. It consists in the main of five articles presented at the Congress by Dr. Kenneth G. McKenzie, Professor of Surgery at the University of Toronto, one of them in conjunction with Dr. Eben Alexander, junior. The subjects of these articles are intracranial astrocytomata, acoustic neuroma, surgical treatment of spasmodic torticollis, Ménière's syndrome and trigeminal tractotomy. Two of the articles are followed by an account of the subsequent discussion. The remainder of the book is made up of two symposia: one is on cervical trauma, presented from the orthopædic, neuro-surgical, rehabilitation and medico-legal aspects; the other is on medico-legal aspects of head injury,

with contributions on electroencephalography and head injury, the legal aspects of epilepsy, rehabilitation and compensation, and neurosurgical considerations. The volume is attractively produced and should be most acceptable to those with interest in the special fields concerned.

Chlorpromazine and Mental Health: Proceedings of the Symposium Held Under the Auspices of Smith, Kline and French Laboratories, June 6, 1955, Warwick Hotel, Philadelphia, Pennsylvania; 1955. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 200, with illustrations. Price: £1 12s. 3d.

THIS volume records the proceedings of a symposium held under the auspices of Smith, Kline and French Laboratories in Philadelphia on June 6, 1955. It includes a series of papers on the use of chlorpromazine by leading psychiatrists with a record of formal and informal discussions which follow. The four main papers were entitled "The Effect of Chlorpromazine on Severe Mental and Emotional Disturbances", "The Immediate and Long Range Effects of Chlorpromazine on the Mental Hospital", "The Effect of Chlorpromazine on Moderate and Mild Mental and Emotional Disturbances", and "Medical and Other Measures to be Taken to Keep the Discharged Patient from Returning to the Institution". The volume will be received with interest by all who are concerned in the use of this significant new drug in the psychiatric field.

"The Compend": A Compendium of Ethical and Proprietaries Used in Medicine and Pharmacy, by William Hetherington, F.P.S.; 1955. Bristol: John Wright and Sons, Limited. 7¼" × 4", pp. 706. Price: 32s. 6d.

OVER 2500 proprietary preparations are listed in this conveniently sized book. A monograph on each of these preparations gives the name of the product, relevant poison schedules if any (this of course applies to the United Kingdom), the name of the manufacturer or distributor, the composition, the form in which it is presented, therapeutic indications, dosage, mode of administration, contraindications where applicable or cautionary notes where necessary, and the packs in which the article is supplied. The information has been derived from that provided by the manufacturers in their descriptive literature and is presented without comment by the compiler of the book, who is a pharmacist. The book should therefore be regarded not in any sense as a text-book of therapeutics, but simply as what it sets out to be—an easily consulted and comprehensive guide to proprietary drugs. Its usefulness is considerably increased by the inclusion of a list of "approved names", descriptions of international units and standard values, a "guide to monographs", by means of which one can find a proprietary preparation when only its basic drug is known, a therapeutic index, which is wisely not ambitious, and a list of manufacturers and distributors.

A.M.A. Scientific Exhibits, 1955, sponsored by Council on Scientific Assembly, American Medical Association; 1955. New York: Grune and Stratton. 11" × 8", pp. 808, with numerous illustrations. Price: $20.00.

EACH year at its annual meeting the American Medical Association presents what are known as the Scientific Exhibits. These are essentially visual presentations by means of photographs, charts, diagrams, pathology specimens, instruments and so on, setting out the latest medical discoveries, experiments and ideas of doctors and scientists. These exhibits have been very popular; so for the benefit of a much wider audience the outstanding material (approximately one-third) of that presented at the 1955 meeting has been collected in this volume. The material ranges over the whole field of medicine, including general practice and most of the specialties, and should have a wide appeal.

Cardiovascular Surgery, by Gerald H. Pratt, M.D., F.A.C.S.; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6", pp. 844, with 358 illustrations. Price: £8 1s. 3d.

THE symposium of which this volume records the proceedings was remarkable for the comprehensiveness of its material (within the limits of the main subject) and the eminence of its participants. The list of official participants numbers 60, made up of leading physicians and surgeons from many parts of the world, and some 478 doctors attended the meetings. The papers presented and the subsequent discussion are here published in full in a handsome well illustrated volume. Thirty-one papers and reports of panel discussions are included, grouped under the following main subject headings: diagnostic techniques, pulmonic stenosis, adjustments between the systemic and pulmonary circulations, the evaluation of transposition operations, mitral stenosis and mitral insufficiency, aortic stenosis and aortic insufficiency, septal defects, cardiac arrest, hypothermia, aneurysms and occlusive diseases of the aorta. This volume, despite its price, will be attractive to all who are interested in this important field which belongs to the whole team of physicians, surgeons and anæsthetists. The brilliant speech made by Sir Russell Brock at the dinner meeting of the symposium has been referred to elsewhere in this journal in a leading article.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Radiology Physics: An Introductory Course for Radiologists, Radiographers and Medical Students", by John Kellock Robertson, M.A., LL.D., F.R.S.C., F.Inst.P.; 1956. London: Macmillan and Company, Limited. 8½" × 5½", pp. 346, with illustrations. Price: 56s. 6d.

The first edition was published in 1940.

"The Royal Northern Hospital, 1856-1956: The Story of a Hundred Years' Work in North London", by Eric C. O. Jewesbury, M.A., D.M. (Oxon.), M.R.C.P., with a foreword by H.R.H. The Duke of Gloucester, K.G.; 1956. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 169, with illustrations. Price: 17s. 6d.

Based on material gathered from minute books *et cetera*.

"Annual Review of Medicine", by David A. Rytand, Editor, and William Creger, Associate Editor; Volume VII; 1956. California: Annual Reviews, Incorporated. 6" × 9", pp. 619. Price: $7.50.

There are 26 chapters to this volume.

"The Year Book of Orthopedics and Traumatic Surgery (1955-1956 Year Book Series)", edited by Edward L. Compere, M.C., F.A.C.S., F.I.C.S.; 1956. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 333, with many illustrations. Price: $6.50.

One of the "Practical Medicine Series" of Year Books.

"The Doctor in Personal Injury Cases", by Harold A. Liebenson; 1956. Chicago: The Year Book Publishers, Incorporated. 4½" × 7", pp. 131. Price: $4.00.

Written by a lawyer in an attempt to be of assistance to medical practitioners.

"Charles Dickens and His Family: A Sympathetic Study", by W. H. Bowen; 1956. Privately printed by W. Heffer and Sons, Limited, Cambridge. 8½" × 5½", pp. 195.

An inquiry into the medical and marital history of the novelist.

"Cardiac Pressures and Pulses: A Manual of Right and Left Heart Catheterization", by Aldo A. Luisada, M.D., and Chi Kong Liu, M.D.; 1956. New York, London: Grune and Stratton, Incorporated. 7" × 10", pp. 123, with illustrations. Price: $6.00.

The nature of the book is implicit in the title.

"Gynecologic Cancer", by James A. Corscaden, Ph.B., M.D.; Second Edition; 1956. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 553, with illustrations. Price: £5 10s.

The author wishes to stress the emotional rather than the technical aspect as the major problem in the treatment of gynæcological cancer.

"A Primer of Freudian Psychology", by Calvin S. Hall; 1956. London: George Allen and Unwin, Limited. 5½" × 8½", pp. 149. Price: 13s. 6d.

The author's object is to rescue Freud "from the domain of mental disorders and to restore him to his legitimate place within the province of normal psychology".

The Medical Journal of Australia

SATURDAY, SEPTEMBER 1, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE UPSURGE OF THE SAVAGE.

RESIDENTS in the more primitive parts of Australia where aborigines are to be found have frequently described their experiences when endeavouring to train an intelligent native girl and bring her up in the ways of white civilization. Often the girl in "preteen age" excels her white school-fellows in quick perception and retentive memory; her sponsors are delighted with her progress and predict for her a future of usefulness. But when she has entered the "teenage" the girl becomes restless, especially if any of her people are camping near by, and then one day she disappears without a word of farewell. When next seen, if she is seen at all, she is a scarred and verminous savage with pendent breasts nursing a baby. The habits of her white sponsors have been shed as a snake sheds its skin; she is happier in her unkempt condition, and any return to the ways of her former life as a foster child is not only impossible but is not desired. We speak with pity of such a reversion and are apt to regard it as a characteristic of the aboriginal and as something from which we are happily exempt, but let us not be too certain and complacent. Mankind has for so many tens of thousands of years indulged in wild magical rites and orgies of sexual licence, with stamping of feet to the rhythm of tom-toms, frenzied dancing of warriors and witch-doctors, with gashing of skin and enforcement of hideous mutilations, that even in the best of us there are interwoven in our psychic background the predispositions and sinister potentialities of the savage. Wordsworth wrote that heaven lies around us in our infancy, an aphorism which a mother of a healthy family of young children is hardly likely to accept as true; but Wordsworth proceeds to deplore the fact that shades of the prison house begin to close about the growing boy. Puberty, especially in the boy, not only witnesses the onset of sexual desire and gratification, but a host of turbulent and often antisocial impulses. It is just at this age when he needs vigilant watching and careful guidance that he is too often pushed out into the world to earn a wage and pick up any contamination offered in his environment. We are horrified at the extent and nature of vandalism; upholstery of railway cars is slashed, young trees, carefully chosen and planted, are uprooted or cut down, sacred memorials are desecrated, devices designed for young people's own protection are wrecked and too often acts of sabotage are committed which in a high degree endanger human life, as when obstructions are placed on railway lines. We are often told that education is an asset which is permanent, it cannot be lost or squandered, it cannot be stolen by thieves or filched by cunning confidence tricksters; but, alas, this is not quite true, for examination of recruits for army or industrial personnel often reveals strange gaps in knowledge—bad spelling, bad grammar, gross ignorance of history and utter lack of interest in the important doings of the day. Those who deplore such findings are apt to attack local systems of education as grossly incompetent, but this is unfair; the education offered is of high grade, but, like the civilized behaviour of the aboriginal girl brought up in a white home, it is overwhelmed by the upsurge of savagery innate in the growing boy and girl of all races and liable to take command of character unless deftly held in check. We should keep this consideration always in mind. We read of rock and roll dances in which young folk are carried away by an hysterical frenzy and behave as if demented; they are merely giving way to elemental forces deeply implanted in human nature. Adults simply cannot understand the screaming adulation of crooners and similar entertainers given by silly girls, but these are only reverting to ancestral usages. We learn from the Bible that the Hebrews in their migration to the Land of Promise, despite evidence of Divine protection, broke away from the austere monotheism which has been the great gift of the Semite to humanity, and as soon as Moses's back was turned, united in crazy worship of the golden calf. This was no new invention, but simply a revival of a primitive idolatrous ritual.

Our daily life displays evidences of magic all around us. The mascot, whether a living animal or an inanimate object, is accepted as something giving a human touch to the actions of the possessor, but is in fact a piece of pure mumbo-jumbo. Revealing of the future by divination is observable in lucky numbers and in tips for horse-racing given by haphazard occurrences and coincidences. Around each lottery there has grown a huge system of unwarranted optimistic beliefs and hopes which lure the prospective buyer of tickets; the hard facts of mathematical theory of probability do not fit in with aspirations based on magical premises.

The great service of Hippocrates to medical science was the divorce of medicine from the supernatural, but, alas, that did not persist as a guiding principle. Soon magic reentered medical practice and remained until nineteenth century science accepted the challenge and restored the Hippocratic tradition. That is to say, in the ranks of the medical profession magic no longer exerts any influence, but unfortunately this is not true of the lay public which, as the old Latin adage tells us, "likes to be deceived". It

is a sad fact that the anxious patient would sometimes prefer a bit of hocus-pocus in the attention of his family doctor. Often he will renounce the trained medical man and seek comfort in the advice and irrational ritual of the herbalist, faith-healer and quack. To the medical practitioner who is doing his best for his patient this attitude can be very irritating, but it should be remembered that it is merely another instance of the savage within us bursting through the logical confines in which scientific progress has enclosed us. Sooner or later science will win and magic be forced into dark corners; it may take longer than we expect as the powers of darkness are still mighty in human nature. The great practical issue is that not only must we offer good education to boys and girls, but that we should be conscious of this urge to let the elemental savage within us take over the direction of our lives. Precept and example, opportunities for work and healthy recreation are to be consistently offered, whilst in the case of the boy and youth it would be wise if we did not allow reaction against the severe corporal punishments in past times to lead us to the opposite extreme.

Current Comment.

NATURAL IMMUNITY.

THE mechanism by which the normal body resists invasion by microorganisms has long been a subject of discussion and experiment. Over fifty years ago Metclinikoff stressed the importance of phagocytic cells, such as the polymorphonuclear leucocytes, in protecting animals from infectious disease. But there seemed to be a plasma component, too, which aided in the destruction of invading organisms. The nature of this was not known, but in most cases it was not a specific antibody such as is produced in immune reactions.

In 1954 L. Pillemer[1] and his associates described the method of isolation of a new serum protein, properdin, which seemed to have an important role in resistance to infection. This protein was found to be active against certain bacteria, viruses and protozoan parasites. It also interacts with a complex polysaccharide called zymosan which forms the outer wall of yeast cells and with a variety of complex polysaccharides of varying origin. A long series of papers on various aspects of the activity of properdin has since appeared from the laboratory of L. Pillemer. The last of these papers by A. C. Wardlaw and L. Pillemer[2] deals with the bactericidal activity of the properdin system and sums up what is now known of the system. The constituents of the properdin system are the protein properdin and the four components of complement and magnesium, and all of these are necessary for any of the activities of properdin. Properdin is quite distinct from antibody in many respects. Zymosan, the insoluble cell wall residue of yeast, combines with properdin in the presence of the four components of complement and magnesium, and during the process the third component of complement is destroyed. Antigen-antibody aggregates inactivate C'1, C'2 and C'4 of complement and spare C'3. The reaction of properdin with zymosan can be used for the concentration of properdin up to 7000-fold.

The present paper deals with the activity of the properdin system against a large range of bacteria. In all the bacterial groups tested some strains were highly susceptible to the system, others resistant and others doubtful. Each strain gave consistent results when tested against a variety of samples of serum. It is suggested that the resistant strains might react under different conditions such as might be found in the body. Since properdin reacts with a variety of complex polysaccharides and zymosan and the outer walls of bacteria consist of complex polysaccharides, the properdin system may, chemically, be relatively specific. Removal of the properdin from serum removes the bactericidal activity of the serum and addition of properdin to such serum restores its bactericidal activity. Properdin has been found in human serum and the sera of a wide range of mammals. If one accepts the findings of Pillemer and his associates, properdin would seem to be an important blood constituent in the prevention of infection. Although they have published fifteen papers on various aspects, no other investigator seems to have published anything, and one would feel much happier if their results could be confirmed in an independent laboratory.

From the Rockefeller Institute of Medical Research has appeared a paper by J. G. Hirsch[1] on phagocytin, a bactericidal substance from polymorphonuclear leucocytes. Although it has long been known that these leucocytes phagocytose microorganisms and destroy them, the mechanism for their destruction has not been clear. Lyozyme is the only bactericidal enzyme so far found in leucocytes, but leucocytes destroy bacteria which are not affected by lyozyme. Hirsch has perfected a method for obtaining large quantities of practically pure polymorphonuclear leucocytes from rabbits, and this material has been used to determine the bactericidal activity of leucocytes and for preparing phagocytin. The leucocytes are disrupted by physical means and extracted with aqueous salt solutions. From these extracts come concentrated solutions of a substance which manifests striking bactericidal activity, especially on Gram-negative enteric bacilli. The organisms are killed, but not lysed. Extracts of human and guinea-pig microphages show less bactericidal activity than rabbit cell preparations. Similar extracts of rat and mouse polymorphonuclear leucocytes contain no demonstrable phagocytin, which is peculiar when one considers the extraordinary resistance to infection shown by rats. Phagocytin is also found, in smaller amount than in the leucocytes, in rabbit liver and spleen. It appears to be a protein with general properties characteristic of a globulin, but is very unstable and has not yet been isolated. The bactericidal activity of phagocytin on Gram-negative enteric bacilli is influenced by the reaction of the medium; the more acid the environment, the more marked is the activity. It has long been known that the pH of the cytoplasm about phagocyted particles is quite low, probably about pH 4·5, at which phagocytin is particularly active. The finding of these two substances, properdin and phagocytin, in mammalian blood is interesting, but there is not sufficient evidence yet to say what part they play in natural non-antibody immunity to infection.

[1] *Science*, August 10, 1954.
[2] *J. Exper. Med.*, May, 1956.

PSYCHIATRIC APPROACH TO GASTRO-INTESTINAL DISEASES.

THE role of the mind and of mental stress in the causation of somatic disease is nowhere more apparent than in a study of gastro-intestinal abnormalities. The exact importance of the psychosomatic element is unknown, and though treatment for diseases such as peptic ulceration and ulcerative colitis is to many people primarily surgical, there is a good deal of evidence that either psychotherapy, or the removal of the patient from the stressful environment, may be of great importance in the prevention, and in the cure, of severe gastro-intestinal diseases. However, psychosomatic medicine is still in its early stages, and the reports of treating gastro-intestinal diseases by psychiatric means are relatively few. A. D. Weisman[1] has reported a special study of six patients suffering from exacerbations of ulceration of the duodenum. Each patient was interviewed at least 25 times and the treatments by psychoanalysis were long. In each patient the basic "effects" were restrained resentment, angry guilt, and guilty fear.

[1] *J. Exper. Med.*, May, 1956.
[2] *Psychosom. Med.*, January-February, 1956.

Recurrence of ulcer symptoms invariably occurred at a time when the subject was vacillating between the opposite tendencies of his emotional pattern. The ego ideal was excessive and fundamentally the underlying motivation of these patients was for the right to be passively loved with exclusive devotion. Symptoms occurred most often when the patient had to restrain the anger caused by threats to the balance of depletion and replenishment. However, Weisman, in discussing the difficulties of psychotherapy in these patients, suggests that further work is needed before the value of the procedure in maintaining the symptom-free interval can be established.

The value of chlorpromazine in the induction of a state of well-being, and in the relief of acute anxiety disorders, is now well established, and, logically, trial has been made of the effect of this drug in the psychosomatic diseases of the gastro-intestinal tract. If the action of chlorpromazine is to suppress the patient's awareness and his exaggerated response to adverse stimuli of a physical or emotional type, then, on the basis of Weisman's explanation of psychosomatic disease, the drug should be useful in controlling these psychosomatic complaints.

L. M. Asher[1] has treated 62 patients, each suffering from severe gastro-intestinal disease of primarily psychosomatic origin, though in several cases there was severe organic involvement. In each instance previous therapy had failed. No regular attempt was made to assess the healing rate of peptic ulcers by radiological means, and observations were based primarily on the patient's report of his own subjective sensations. Some control was exercised by the occasional substitution of placebos for the treatment. The average dosage of chlorpromazine was 25 milligrammes three times a day for from two weeks to six months. The only noted initial side effect was some drowsiness. Forty-one patients responded favourably, mostly those in whom the digestive disturbance was related to anxiety. In each of the five cases of duodenal ulcer, there was considerable improvement, especially in the accessibility of the patients. The two patients with gastric ulceration were promptly relieved of pain, and in one there was radiological evidence of healing of the ulcer. Of the 21 patients with an anxiety state and associated indigestion without organic signs, improvement occurred in 19, and it was especially favourable in the younger patients. Substitution of the placebo was soon followed by a return of symptoms in these patients. Improvements were noted in a whole variety of gastro-intestinal complaints. Complications of therapy included exaggeration of gastric irritability or diarrhœa, postural dizziness, skin rashes, and in one case a temporary agranulocytosis, and one possibly drug-induced hepatitis.

Failures of treatment were encountered in one way or another, and these were partially due to the reluctance of the patient to part with the illness. In this respect failure was particularly noticeable in patients with chronic ulcerative colitis, hysterical distension and depression with constipation and flatulence. Most other conditions responded favourably; in fact in one patient the correct diagnosis of carcinoma of the head of the pancreas was masked for some months.

The treatment of ulcerative colitis has always been a difficult and very disappointing field of work. While a multitude of different modes of therapy exist, it is hard to be sure that the occasional cures are no more than a remission in this unpredictable complaint. The surgical approach, while often the most satisfactory, literally avoids the primary problem by by-passing it. Psychotherapy has been little more successful and the results of chlorpromazine from the report of Asher appear to be very disappointing. The distress caused by chronic ulcerative colitis to patients, relatives and medical attendants alike may well induce the impression that even the most heroic measures are worth trying. Occasional reports have been made of single cases successfully treated by prefrontal lobotomy. R. W. Levy, H. Wilkins, J. D. Herrmann, A. C. Lisle and A. Rix[2] have reported on the use of this procedure in five patients with chronic ulcerative colitis. Each patient had failed to respond to medical and psychiatric treatment, and each was considered to be in too poor a physical state to undergo colectomy. In three of the patients after the lobotomy there was considerable improvement, with healing of ulcers and a change towards the normal in the radiological appearance of the large bowel, so that the patients were able to lead normal lives. In a fourth patient the effect of lobotomy was temporary, as was also a subsequent extensive bilateral prefrontal lobotomy, and the patient died after a final colectomy. The fifth patient died one week after operation, when he developed an obstruction of the large intestine followed by perforation. The first death could hardly be attributable to the neurosurgical technique one year previously, and represents a failure of lobotomy rather than a fatality. The second death possibly represents the danger of throwing undue load on the gastro-intestinal tract before healing of the colitis has taken place. On the whole, despite the complications, the report of this series is encouraging, and might well hold some hope for patients suffering from chronic ulcerative colitis, than whom there can be few more miserable.

[1] *J.A.M.A.*, April 14, 1956.
[2] *J.A.M.A.*, April 14, 1956.

KIMMELSTIEL–WILSON SYNDROME.

In 1936 a paper by P. Kimmelstiel and C. Wilson[1] described a renal lesion which was one of striking hyaline thickening of the intercapillary connective tissue of the glomerulus. The characteristic clinical features were a previous history of *diabetes mellitus*, severe and widespread œdema of the nephrotic type, and gross albuminuria. Hypertension was frequently present and many of the subjects had been found also to have renal decompensation. The renal lesion was said by Kimmelstiel and Wilson to be an intercapillary glomerulosclerosis differentiated from intercapillary glomerulonephritis by the finding, in the latter, of changes in the capillary basement membrane.

G. V. Mann, C. Gardner and H. F. Root[2] added to the accumulating knowledge of intercapillary glomerulosclerosis by studying a series of diabetic patients with disease of the urinary tract. They described the characteristic syndrome of proteinuria, œdema, hypertension, and retinitis, occurring in young people with diabetes of long duration. Death was the result of myocardial infarction, of congestive failure, or of renal failure and uræmia. These authors suggested that prevention of the distinct renal changes could be secured only by better control of the diabetic state. However, there was no general agreement on the clinical syndrome which was likely to lead to the finding of the intercapillary glomerulosclerosis at the post-mortem examination of diabetic subjects. In a series of 24 diabetic patients in whom death was preceded by progressive renal complications, W. R. Gauld, A. L. Stalker and A. Lyall[3] reported that the typical Kimmelstiel-Wilson changes in the kidney were found in subjects who were, with one exception, over fifty years of age, and in whom the diabetic state was not particularly severe. Hypertension was usually severe, appeared after the *diabetes mellitus*, and was followed by retinal changes. Albuminuria and renal failure were noted several years before death.

However, the correlation of clinical findings and autopsy findings in a series of 229 diabetic subjects reported by J. Rogers and S. L. Robbins[4] did not lend support to the view that the Kimmelstiel-Wilson changes in the kidney were related to a particular type of renal failure occurring in diabetic patients. The so-called classical features of intercapillary glomerulosclerosis were present in patients without the anatomical lesion of the syndrome, and the pathological changes were present in patients without the clinical syndrome. In a further paper, Rogers and Robbins,

[1] *Am. J. Path.*, January, 1936.
[2] *Am. J. Med.*, July, 1949.
[3] *Brit. M. J.*, July 24, 1948.
[4] *Am. J. Med.*, June, 1952.

together with H. Jeghers,[1] compared the clinical signs of 100 diabetic patients with intercapillary glomerulosclerosis and 176 diabetics with no such renal changes. Though no fine differences were noted between the two groups, there were definite tendencies. The typical renal changes occurred more frequently when the history of *diabetes mellitus* was long, and were found in 64% of patients in whom the diabetes was of ten to fourteen years' duration. Also, patients with intercapillary glomerulosclerosis had tended to have less glycosuria than would have been expected from the degree of hyperglycæmia. Hypertension, albuminuria, azotæmia and cardiac failure were all more frequent in patients with the Kimmelstiel-Wilson changes. Uræmia, cardiac decompensation and cerebro-vascular accidents were each significantly more prevalent in the patients with intercapillary glomerulosclerosis.

The post-mortem examination of 43 diabetic subjects led I. Gilland[2] to suggest that the Kimmelstiel-Wilson group comprised, especially, elderly females who had had mild diabetes for many years. The patients with the syndrome had, in addition to the non-specific proteinuria of renal disease, hypertension with heart failure, peripheral neuropathy, œdema at least of the dependent parts, and retinitis or cataract obscuring the retina. Gilland suggested that any patient who had these four clinical features, together with proteinuria, was likely to have Kimmelstiel-Wilson lesions in the kidney.

G. S. Anderson[3] separates the Kimmelstiel-Wilson lesions of the kidney into nodular and exudative forms, the latter being the less common. He has noted several additional features of the histology of the lesions, and suggests that the lesions are part of a common process which is that of the deposition of fibrin or other protein substance on endothelium. Anderson believes that nodular glomerulosclerosis is the result of the plugging of the capillaries with fibrin or some other protein coagulum. The exudative form is probably a terminal event.

H. P. Taft, E. S. Finckh and R. A. Joske[4] have studied a series of living diabetic subjects by the examination of material obtained in biopsy of the kidneys. No patient had the full clinical form of the Kimmelstiel-Wilson syndrome, but in several there were pathological changes in the glomeruli. The changes varied from acidophilic material in or between the capillary basement membranes, to the more usually described intercapillary lesions of the syndrome. Occasionally, glomeruli were converted into a fibrous ball, identical with those of the sclerosed glomeruli of ischæmia, found in senile nephrosclerosis. Nodular glomerular changes were not found in young diabetic patients; pathological lesions were generally more severe in the older patients and in those with more long-standing diabetes. The changes were not apparently influenced by the effective control of the level of the blood sugar.

The most recent attempt to correlate the findings of the Kimmelstiel-Wilson syndrome with the progress of the *diabetes mellitus* has been made by F. H. Epstein and V. J. Zupa.[5] These authors have reviewed the records and perused the sections of kidney obtained from 137 diabetic patients examined *post mortem* at the New Haven Hospital, Connecticut, between 1944 and 1953. Of the subjects, 37 were found to have the nodular glomerular lesions of Kimmelstiel and Wilson. In the comparisons, a series of 37 similar diabetic patients with no nodular glomerulosclerosis were used. Arteriosclerosis of the larger renal vessels, as well as arteriosclerosis of varying degree and extent, was present in every case of the Kimmelstiel-Wilson syndrome. Twenty-two of the 37 also had, at some time, an elevation of the level of non-protein nitrogen. In the control group, arteriosclerosis was present in 23 and prominent in seven, while in only three was there an elevation of the level of non-protein nitrogen. The incidence of pyelonephritis was much less in the control group. The age and sex distribution was not significantly different in the two groups. Particularly associated with the Kimmelstiel-Wilson group were persistent albuminuria, hypertension, neuropathy, diabetic retinopathy, and low levels of blood albumin. Both dependent œdema and congestive heart failure were very frequent amongst the patients with intercapillary glomerulosclerosis. No correlation with the necessary insulin dosage was noted. The frequency of diabetic acidosis in patients with the kidney syndrome did not differ from that observed in diabetic patients without this lesion. The findings in this series do not differ significantly from most other attempts to identify the clinical characteristics of the Kimmelstiel-Wilson syndrome. However, there is no support for the view that the renal changes are associated with a specific type of *diabetes mellitus* characterized by infrequent episodes of acidosis and decreasing requirements of insulin.

[1] *Am. J. Med.*, June, 1952.
[2] *Brit. M. J.*, April 28, 1951.
[3] *J. Path. & Bact.*, January, 1954.
[4] *Australasian Ann. Med.*, August, 1954.
[5] *New England J. Med.*, May 10, 1956.

INFANTILE GASTRO-ENTERITIS.

GASTRO-ENTERITIS, once one of the leading causes of infantile deaths in Australia, is now relatively unimportant; the most recent death rates are less than 4% of what they were in the first decade of this century (H. O. Lancaster[1]). Despite this spectacular decline, outbreaks of gastro-enteritis occur from time to time in infants and young children. A number of these have been investigated, and from the bacteriological findings it is apparent that the causal organism in the majority of these epidemics is a strain of either Shigella or Salmonella (D. C. Fison,[2] S. Williams,[3] R. Mushin,[4] V. L. Collins[5]). Another type of epidemic, one which occurs among infants in institutions or hospitals, has received considerable attention overseas. The significant fact about these epidemics is that bacteriologically they have been shown to be caused by one of a group of particular types of *Escherichia coli* (*Bacterium coli*), which are pathogenic for infants, whereas they are relatively harmless in the gastro-intestinal tract of adults and older children.

Recently, J. Smith,[6] Lister Fellow of the Royal College of Physicians of Edinburgh, has surveyed the now extensive literature on these strains of *Escherichia coli* and has, in addition, given details of an epidemic which occurred in Aberdeen. The strains of *E. coli* are now classified according to the method of F. Kaufmann,[7] which is based on (*a*) the biochemical characteristics, and (*b*) the serological behaviour of these organisms. The identification is a long and tedious business and could hardly be made as a routine procedure in a hospital or public health laboratory. Most of the investigations have been made by special units set up for the purpose. Up to the present 12 types of *E. coli* have been incriminated in different parts of the world as the causal organisms of epidemics. In the Kaufmann classification they are O111, O55, O26, O86, O125, O126, O128, O119, O25, O44, O127, O114. There appear to be a number of subtypes of some of these. The order in which the types are listed corresponds to the frequency with which they have been incriminated in epidemics. In part this difference may reflect both the length of time for which the type has been known and the diligence of the search for other strains in "non-specific" epidemics of gastro-enteritis.

The difficulty of identification may explain, in part, why there have been so few investigations in Australia aimed at the incrimination of specific strains of *E. coli* as causal organisms of epidemics. However, *E. coli* O111 has been identified as the probable ætiological factor in epidemics of

[1] M. J. AUSTRALIA, July 21, 1956.
[2] M. J. AUSTRALIA, December 30, 1950.
[3] M. J. AUSTRALIA, August 4, 1951.
[4] M. J. AUSTRALIA, March 29, 1952.
[5] M. J. AUSTRALIA, October 18, 1952, 570 (discussion at Tenth Session, Australasian Medical Congress (British Medical Association)).
[6] SMITH, J. (1955), "The Ætiology of Epidemic Infantile Gastro-enteritis". Edinburgh: The Royal College of Physicians. 8¾" × 5½", pp. 104.
[7] *J. Immunol.*, September, 1947.

gastro-enteritis on at least four occasions. Williams in Sydney in 1950-1951 recovered the organism from 24 children, 13 of whom were diagnosed as suffering from "non-specific" enteritis; three also had a Salmonella infection; and seven were free from gastro-intestinal symptoms. Mushin summarized the Melbourne experience to 1952, showing that *E. coli* O111 has apparently been recovered from a number of children with gastro-enteritis. E. Singer and C. G. Ludford[1] made an extensive investigation of an outbreak of gastro-enteritis in an infants' home in Brisbane and recovered *E. coli* O111 from 11 children. They remark that the organism had only recently arrived in Brisbane. It is interesting to record that no epidemic of gastro-enteritis which might be reasonably thought to be due to a specific type of *E. coli* has occurred in an infants' institution or hospital in New South Wales since gastro-enteritis became a notifiable disease in July, 1952. In the Aberdeen epidemic the maximum incidence was in the first trimester of life, with a peak at two months of age. Epidemics always occur among collections of infants, and the original source of the infection usually remains obscure. Almost all infants affected are artificially fed, and defective nutrition or previous disease is a predisposing factor. The incubation period in Smith's cases was from two to ten days. In the Brisbane series it is of interest to note that *E. coli* O111 was isolated from children three to five days before clinical signs appeared. The mortality in Smith's series was slightly over 40%; in Brisbane two children died out of 11 from whom *E. coli* O111 was isolated. In the Aberdeen epidemic the onset was sudden in most cases, with vomiting and abnormal stools, and the majority showed from mild to severe dehydration. An interesting feature of the pathological findings is how often the gastro-intestinal tract was devoid of any pathological changes either on microscopic examination of the surface of the gut or on histological examination.

The disease is highly infectious, and a rapid spread to the majority of artificially fed infants is a characteristic of most epidemics. Smith points out that the control of epidemics of gastro-enteritis in infants' wards and institutions presents a problem which demands the best in nursing techniques, for an infant with acute diarrhœa and vomiting due to one of the specific types of *E. coli* excretes vast numbers of organisms capable of giving rise to further infection.

HYPOKINETIC DISEASE.

H. KRAUS, B. PRUDDEN AND K. HIRSCHHORN,[2] in a paper on the role of inactivity in production of disease, state their belief that many orthopædic disabilities and neuromuscular tension syndromes are due to under-exercise. They bring considerable evidence to show that in the United States, owing to the mechanized way of life, over half the population grows up in a state of muscular deficiency. It has been shown several times that the incidence of death from coronary disease is doubled among the physically less active.

In a study on low back symptoms the authors showed that approximately 80% of their patients were free from organic disease, but failed to pass six simple exercise tests for strength and flexibility of key posture muscles. Over 5000 cases of low back pain were studied, and most of the patients lost their symptoms when given a course of corrective exercises. Most of these patients presented a combination of both physical and emotional complaints. An examination of school children brought out some extraordinary results. Approximately 40,000 American children between six and sixteen years of age coming from public school systems in suburban and urban communities were examined. The tests were standardized and were all carried out by the authors themselves. Children from comparable socio-economic groups in Italy, Austria and Switzerland, where different conditions of nutrition and physical education existed, but where life was not particularly mechanized, were similarly examined. There were 2870 of these children. The American children were considerably weaker and much less flexible than the comparable groups of European children. The great difference between the two groups was in the extent to which they used their bodies for locomotion, play and entertainment. These results indicate that American children are brought up without the benefit of the minimum physical activity necessary for healthy living. The authors claim that there will be an increase in hypokinetic diseases such as coronary disease and certain orthopædic difficulties, and also an increase in emotional instability and decline in mental health. How far these conditions apply to Australia cannot be said. Undoubtedly Australian children do exercise much more than comparable American children, but increasing mechanization of living is evident enough in Australia.

The medical profession must in some respects revise its opinion about rest and exercise. Rest is not necessarily a prerequisite for healing; it may well interfere with it. Avoidance of over-use of rest is particularly applicable in elderly persons. Therapeutic exercise should be more commonly used.

[1] *Australasian Ann. Med.*, May, 1953.
[2] *J. Am. Geriatrics Soc.*, May, 1956.

CHRONIC HEADACHE AND THE TEMPORO-MANDIBULAR JOINT.

ALTHOUGH J. B. Costen[1] showed in 1936 that dysfunction of the temporo-mandibular joint is frequently associated with a number of symptoms in the head region, particularly chronic headache, in medical literature very little notice has been taken of his investigations. R. Berlin, L. Dessner and S. Aberg[2] report on an investigation of 105 patients with chronic headaches associated with disordered function of the temporo-mandibular joints. There were local signs around the joints which might be well marked or hardly noticeable, but it is the peripheral discomfort which dominates the picture. The patients complain of a persistent aching pain, usually concentrated to the crown and the temples or the forehead and the temples, but sometimes also the back of the head. There may be pain in other parts of the head. It is usually found that, in connexion with opening and chewing movements, the mandibular joints creak and cause pain and tenderness. X-ray examination is useful for diagnosis, for usually one or both condyles are displaced upwards and backwards in the glenoid fossa when the teeth are clenched. In advanced cases degenerative changes may be seen in the joints. The treatment is fundamentally dental and orthopædic, particularly in the adjustment of the bite. Removable splints are usually necessary and some selective grinding of the teeth. Within a few days after this treatment is started an improvement is usually seen. Very rarely are more drastic measures necessary, such as operation on the joint. The condition is not rare; 105 sufferers were seen in two and a half years in a moderate sized hospital in Sweden. The headache in most of these cases manifested itself a few years after extensive extractions of molar teeth and often after careless prosthesis. The condition can last for a long time—over twenty years in five instances. Of the patients, 83% were completely cured or experienced considerable improvement as a result of simple dental treatment.

The authors discuss the causal factors of the headache. They believe that this special type of headache belongs in the group of complaints caused by overloading and is provoked by disturbances in the masticatory muscles. As the causation of chronic headaches is frequently difficult to find and usual methods of treatment are unsatisfactory, the syndrome discussed here should be borne in mind.

[1] *J.A.M.A.*, July 25, 1936.
[2] *Acta med. scandinav.*, Vol. 54, 1956.

Abstracts from Medical Literature.

DERMATOLOGY.

Mepacrine and Chloroquine in the Treatment of Rosacea.

H. BRODTHAGEN (*Brit. J. Dermat.*, December, 1955) suggests that since mepacrine, and later chloroquine, proved to be effective in the treatment of *lupus erythematosus*, which is often provoked by sunlight, it was reasonable to investigate the effects of these drugs on other diseases which can be partly or wholly provoked by light. Since the aetiology of rosacea is probably a complex one, it is difficult to investigate one factor alone, in this case provocation by sunlight. This study showed that the group of patients who report exposure to sunlight as a provocative factor can be improved or cured by agents which increase the light tolerance. The dosage was fixed in accordance with experience in the treatment of chronic polymorphic light eruptions, in which the effective dose appears to be 100 milligrammes of mepacrine or 200 milligrammes of chloroquine daily. No case of agranulocytosis or aplastic anaemia occurred. There is less risk of provoking a dermatitis when chloroquine is used. The treatment of rosacea with mepacrine or chloroquine should be reserved for cases in which the light provocation has been definitely ascertained by the patient himself. The susceptibility of the skin to quantitative irradiation does not appear to afford any guidance.

Skin Hazards of Coal Mining.

G. HODGSON (*Brit. J. Dermat.*, December, 1955) considers that the hazards of coal mining seem to be attributable more to the physical conditions of the work than to chemical irritation or sensitization. The physical conditions expose the skin to trauma, coal and stone dust, sweating and wet conditions. The commonest sites of abnormalities of the skin are the hands, lower parts of the legs, feet and ankles, and areas of contact with the lamp battery, belt, axillary clothes, and helmet. Positional working which causes the friction of dirty clothes on the skin seems to be responsible for most of the skin diseases seen in coal miners.

Acneiform Eruption Due to Corticotropin.

M. SULLIVAN AND I. ZELIGMAN (*Arch. Dermat.*, February, 1956) state that some patients who are receiving corticotropin develop an eruption which, in many respects, resembles that of *acne vulgaris*. The histological features of the two conditions and certain clinical features distinguish the drug eruption from that of *acne vulgaris*. There are similarities in the development of the two conditions. The primary alteration is one of hyperkeratinization of the follicular orifice, followed by the formation of an epithelial cyst. Later, there occurs perifollicular infiltration. In some cases a foreign body type of granulation tissue forms and suppuration occurs. The lesion of *acne vulgaris* is larger than that of the drug eruption. Abscess formation is more frequent in *acne vulgaris*. The most important difference lies in the normal appearance of the sebaceous glands in the corticotropin eruption, as contrasted with the hyperplasia of the glands found in *acne vulgaris*. The dual origin of *acne vulgaris* is in the imbalance between the adrenal cortex and the ratio of androgen or progesterone to oestrogens. This contrasts with the single origin of the acneiform eruption in the activities of the adrenal cortex. These different origins may account for the distinguishing gross and microscopic findings.

A Treatment for Monilial Paronychia.

C. REITER (*Arch. Dermat.*, February, 1956) suggests that in the treatment of monilial paronychia a silicone protective may be applied each morning to protect the fingers from the effect of the frequent immersion in water which seems to be the greatest single factor for the persistence of the paronychia. At night, the following application should be applied to the nail bed: resorcinol 1·5 grammes, acetone 1·0 millilitre, boric acid 1·0 gramme, water to 60 millilitres. This medication is applied with a very fine brush rather than with the standard applicator or orange stick. The use of the brush facilitates the application of the mixture to all the crevices, without the subsequent loosening of the nail fold. With this treatment the authors obtained uniformly good results in cases of monilial paronychia.

Persistent Penile Plaques.

M. SULZBERGER, V. H. WITTEN AND J. A. HUNT (*Arch. Dermat.*, February, 1956) consider that four particular forms of persistent penile plaques are often puzzling. These dermatoses are *lichen planus*, psoriasis, distinctive exudative discoid and lichenoid chronic dermatosis, and erythroplasia of Queyrat. The differential diagnostic features are reviewed in detail. Emphasis is placed upon the difficulty in distinguishing between the precancerous or cancerous erythroplasia of Queyrat and the persistent penile plaque of exudative discoid and lichenoid chronic dermatoses. There is need for careful examination of the whole skin, its appendages and accessible mucous membranes, and for a searching inquiry into the history to discover past or present accompanying lesions which might indicate the true nature of the persistent penile plaque. True erythroplasia occurs almost exclusively in the uncircumcized, and the possible carcinogenic role of smegma is cited.

Perifolliculitis Capitis Abscedens et Suffodiens.

F. H. MCMULLAN AND I. ZELIGMAN (*Arch. Dermat.*, March, 1956) state that *perifolliculitis capitis abscedens et suffodiens* is a rare disease which begins as a simple folliculitis of the scalp and soon progresses to develop the appearance of more advanced disease. As in the case of acne, the process is initiated by keratinization and occlusion of the follicular orifice. The staphylococcus is the responsible organism in the infectious component of the disease. Four patients who had received adequate antimicrobial therapy without permanent benefit, were subsequently successfully treated by the temporary epilation induced by X radiations.

Prednisone in the Treatment of Dermatoses.

C. R. REIN AND E. L. BODIAN (*Arch. Dermat.*, April, 1956) have studied the reactions of 41 patients with various dermatoses to the administration of prednisone by mouth, taken in three or four divided doses of from 15 milligrammes to 60 milligrammes daily. In this small series of patients the authors were favourably impressed with the therapeutic efficacy of prednisone, which seemed to be four to five times more potent than hydrocortisone or cortisone. The initial therapeutic response in the chronic dermatoses appeared to be slower with prednisone than with the previously employed steroids. The side effects encountered were minimal. There was no evidence of salt retention, hypertension, sodium depletion, peptic ulcers or glycosuria. Moon facies, however, was not an uncommon side effect, and appeared to develop more rapidly and more frequently than with either cortisone or hydrocortisone. Other side effects included increased appetite with weight gain, gastric distress, menstrual disturbance, acne, hot flushes, increased sweating, headache, vertigo, weakness, depression and insomnia. Most of these side effects disappeared with reduction from the initial to the maintenance dosage. Other hormonal side effects usually associated with adrenal steroid therapy might occur with prednisone. Determinations of the frequency of their appearance in relation to the additional potency of this compound would require prolonged observation. The authors conclude that prednisone is a valuable and therapeutically effective tool to be added to the armamentarium of the dermatologist.

Laboratory Studies in Systemic Lupus Erythematosus.

S. L. LEE (*Arch. Dermat.*, April, 1956) states that a pattern of altered blood proteins can be recognized in systemic *lupus erythematosus*. Four anomalous activities occur, all in the γ globulin fraction. One is responsible for false positive serological findings for syphilis, one for positive direct antiglobulin (Coombs) findings, one acts as an anticoagulant, and the fourth is the *lupus erythematosus* cell factor. The mechanism of the *lupus erythematosus* cell phenomenon and its possible relationship to local and constitutional lesions in *lupus erythematosus* have been discussed. The nature and ultimate significance of the dysproteinaemia of systemic *lupus erythematosus* are unknown.

Polymorphic Light Eruptions Treated with Chloroquine.

J. B. CHRISTIANSEN AND H. BRODTHAGEN (*Brit. J. Dermat.*, June, 1956) have investigated the treatment of polymorphic light eruptions of the skin

with the drug chloroquine, which does not have the embarrassing side effect of yellow skin coloration seen with mepacrine. Patients were exposed to experimental radiation in order to assess changes in the reaction of the skin to ultra-violet radiation during the treatment. The usual treatment of acute eruptions was with 250 milligrammes of chloroquine diphosphate per day, though as much as 1·0 gramme a day was given if this dosage proved to be insufficient. During the period of treatment the patients were encouraged to go out freely, irrespective of the state of the weather, light or shade. The adaptability of the skin to ultra-violet radiation during chloroquine therapy was determined by estimating the minimal erythema dose before, during, and at least three weeks after discontinuation of treatment. This treatment was extended to 58 patients, of whom six experienced unpleasant blurring of vision, sometimes accompanied by disturbances in accommodation. These side effects were overcome by reducing the dosage or by suspending the treatment for a short period. Four patients complained of dyspepsia, but in only one case was this so severe that treatment had to be suspended. The study of 39 patients revealed that the minimal erythema dose was raised from one to two minutes before the treatment to eight to sixteen minutes during the treatment, and the effects persisted to a considerable degree for at least three weeks after treatment had been suspended. Of the 58 patients treated, the majority remained free from symptoms. Of the remainder, most showed a favourable response. It is suggested that chloroquine exerts no direct influence on light tolerance, but can restore the adaptability of the skin through ultra-violet radiation.

UROLOGY.

Wilms Tumours.

M. J. FEENEY, R. B. MULLENIX, R. J. PRENTISS AND J. M. WHISENAND (*J. Urol.*, September, 1955) report on a series of 14 cases of Wilms tumours including three in patients who had bilateral tumours. They state that the multiplicity of primary tumours demonstrates the possibility of multicentric origin. The ages of the patients ranged from three months to nine years. There were six females and eight males. One patient is alive and well fifteen years after nephrectomy. One is alive seven years later, but has had excision of two new extraperitoneal masses. Two patients have been followed for less than a year. The other nine patients died early. The treatment is prompt and radical nephrotomy, followed by deep irradiation.

Treatment of Genito-Urinary Tuberculosis.

J. K. LATIMER (*J. Urol.*, September, 1955) publishes the results of treatment of urinary and genital tuberculosis in 625 patients over a period of seven years. As a result of these studies, and largely because of the changed outlook resulting from combined chemotherapy, the author states that there has been improved prognostic feeling about this formerly lethal disease. In the United States of America genito-urinary tuberculosis occurs as a complication in about 4% of all patients with pulmonary tuberculosis. Tuberculosis sometimes destroys a kidney rapidly (three years), but usually it takes a long time (up to ten years). Some people still die of renal tuberculosis, despite the new drugs. If the renal disease lasts long enough, the bacilli passing down the ureter will infect the bladder. It has been estimated that the average time between the original pulmonary infection and the appearance of vesical irritability is something in excess of eight years. Therefore, all clinicians who attend patients with pulmonary tuberculosis should watch carefully over ten years for pyuria or vesical symptoms. Bacilli tend to drop into the prostatic ducts from the posterior part of the urethra, thus infecting the prostate and later the seminal vesicles. Next they may travel down the *ductus deferens* to the epididymis. About one-half of the patients in the author's clinics had epididymal tuberculosis as the presenting symptom. The important anti-tuberculosis drugs are streptomycin, PAS and "Isoniazid". No one of these should ever be used singly lest drug resistance develop. At present it is thought best to use all three together, and the dosage is one gramme of streptomycin twice weekly *plus* sodium PAS five grammes eight-hourly and "Isoniazid" 100 milligrammes eight-hourly. If a patient cannot tolerate PAS, the other two drugs may be used with very good results. For elderly patients or for those who already have some vestibular or nerve toxic involvement, full treatment with streptomycin may be replaced by a continuation of half streptomycin and half dihydrostreptomycin. An analysis of cases which failed to stay converted from "positive" to "negative" smear findings is informative. The 20% in which there was failure with streptomycin and PAS were in patients who would not stay on bed rest. The author considers that bed rest, or at least semi-bed rest, is helpful during the first six months of treatment. The course is over one year; if any operation is to be performed, it is ideal to precede this by four months of the course, and follow it with the remaining eight months' treatment. Prostatic lesions were converted to "negative" smear findings just as frequently as were the renal lesions; consequently the author and his colleagues have become far more constructive in their treatment of genital tuberculosis. No longer are total prostatectomies performed, and they are slower to carry out epididymectomies. The five-year mortality in patients with bilateral renal tuberculosis receiving chemotherapy is now only 8%. In the years before chemotherapy this mortality was about 80%. Advanced unilateral renal tuberculosis is probably still best treated by nephrectomy, with four months' pre-operative and eight months' post-operative chemotherapy. The object of such a thorough course is to heal any tiny blood-borne tubercles lying dormant in the remaining kidney. The author has performed "partial nephrectomy" (renal resection) in five cases; they still return negative results for tubercle bacilli after two and a half years; chemotherapy is, of course, used as an adjuvant. However, in the past thirty months results with chemotherapy have improved so much that no further operation of renal resection has been indicated. For the patient with bilateral tuberculosis, chemotherapy holds out hope for arrest of the disease; formerly he had no hope at all. But success depends on fairly early treatment; for hollowed out kidneys there is no hope. Contracted tuberculous bladder is a most difficult complication, and people so affected are economic cripples. Diversion of ureters or of a ureter to the skin is far safer than uretero-colonic anastomosis.

Congenital Unilateral Multicystic Kidney.

H. M. SPENCE (*J. Urol.*, December, 1955) has emphasized the features of congenital multicystic kidney. He states that these warrant its classification as a separate entity to be distinguished from congenital polycystic disease, multilocular cysts and multiple simple cysts. True polycystic disease is almost invariably bilateral, and is ultimately fatal; the reverse is the case with multicystic kidney, in which there is an opposite normal organ, and the prognosis after nephrectomy is excellent. The term "unilateral multicystic kidney" was first proposed by Schwartz in 1936. The striking feature of this lesion is replacement of the entire renal parenchyma by many cysts, varying in size from that of a pea to that of a bird's egg, and held together in a form like a bunch of grapes by loose connective tissue. The ureter is characteristically absent or rudimentary. The vascular pedicle may also be rudimentary, or even absent. There is no evidence that the lesion is a variety of hydronephrotic (obstructive) phenomenon. In congenital polycystic disease, varying amounts of normal parenchyma, capable of function, are seen microscopically between the cysts. Multilocular cystic kidney shows the disease process in part of the organ only, and cysts are seen within cysts. The word polycystic should not be used loosely to denote either the multicystic kidney or the multilocular cystic kidney. Polycystic disease is congenital, bilateral and ultimately fatal. As regards the etiology of multicystic kidney, all that can be said for certain is that it is a congenital defect resulting from fœtal maldevelopment, not on an hereditary basis. In true polycystic disease there is, as well as congenitalism, an inherent genetic fault. The author reviews 15 cases previously reported in the literature and describes four cases of his own. He states that on routine examination of a presumably healthy infant, a large, unilateral, non-tender, movable mass is found in one flank. Signs of pressure on the bowel and the symptom of pain are unusual. Cystoscopic examination and attempted retrograde study have shown either absence of a ureteric orifice or blind termination of a small ureter below the affected kidney. Excretion urography shows no function on the affected side. There is no predilection as to side or sex. Most cases occur in infants, but a few are in adults. In the differential diagnosis Wilms tumour and hydronephrosis are to be considered.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the South Australian Branch of the British Medical Association was held at the Verco Theatre, Institute of Medical and Veterinary Science, Frome Road, Adelaide, on June 27, 1956, DR. G. L. BENNETT, the President, in the chair.

MINUTES.

On the motion of Dr. H. A. Goode, seconded by Dr. George Gibson, the minutes of the previous annual meeting were taken as read and signed as correct.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council was received and adopted on the motion of Dr. B. S. Hanson, seconded by Dr. Sholto Douglas. The report is as follows.

At the annual general meeting of members of the Branch held on June 29, 1955, the following officers and members of Council were elected:

President: Dr. G. L. Bennett.

Vice-President: Dr. M. E. Chinner.

Honorary Treasurer: Dr. F. L. Wall.

Honorary Medical Secretary: Dr. Ronald Hunter.

Ordinary Members of Council: Group A, Dr. M. T. Cockburn, Dr. Ian A. Hamilton, Dr. C. C. Jungfer; Group B, nil.

At the first Council meeting of the new year Dr. R. M. Holmes, of Clare, was invited to serve as a group (country) representative on the Council occasioned by the failure to elect, *vide* rule 44. Dr. Holmes accepted the office.

The following subcommittees were appointed:

Scientific: Dr. Hamilton and Dr. Verco.

National Health Service: Dr. Rieger, Dr. Mallen, Dr. Jungfer and Dr. Elix.

Ethics: Dr. Rieger, Dr. Wall, Dr. Verco, Dr. Cockburn, Dr. Chinner and Dr. Jose.

Parliamentary Bills: Dr. Mallen and Dr. Rieger.

Publicity: Dr. Rieger, Dr. Mallen and Dr. Greenlees.

Library: Dr. Cockburn, Dr. Hunter and Dr. Dwyer.

Salaries: Dr. Mallen, Dr. Rieger and Dr. Greenlees.

Medico-Pharmaceutical Liaison Committee: Dr. Elix, Dr. Hunter and Dr. Rieger.

Tuberculosis Standing Committee: Sir Darcy Cowan, Dr. Hayward, Dr. Hetzel, Dr. Sutherland, Dr. Sleeman and Dr. Woodruff.

The President, Immediate Past President (if any), the Vice-President, the Honorary Treasurer and the Honorary Medical Secretary are ex-officio members of all committees other than the Ethics and Standing Committees.

The President and the Honorary Medical Secretary are ex-officio members of the Ethics and of all Standing Committees.

Monthly Scientific Meetings.

Eight scientific meetings have been held during the year as follows:

1955.—July 28: "Abdominal Pain in Children." Dr. C. O. F. Rieger discussed the subject from the surgical aspect, and Dr. Ivan Magarey from the medical point of view. Discussion opened by Dr. Eric Sims. September 29: Clinical meeting at Repatriation General Hospital, Springbank. October 27: "The Prevention of Motor Accidents and the Medical Profession." Opening speaker, Professor J. B. Cleland; guest speakers, Senior Inspector J. J. Turnbull, Mr. F. Campbell (National Safety Council) and Mr. D. S. Dwyer (Royal Automobile Association). Discussion opened by Dr. C. C. Jungfer. November 24: "Obstetrical Emergencies." Speaker, Dr. G. T. Gibson.

1956.—February 23: Clinical evening, Adelaide Children's Hospital. March 28: "Some Complications of Common Fractures." Speaker, Mr. W. J. Betts. Discussion opened by Mr. Lansell Bonnin. April 26: Listerian Oration delivered by Professor Frank Magarey entitled "Some Aspects of Research into Vascular Degeneration". May 31: "Radiotherapy in Benign Conditions." Speaker, Dr. B. S. Hanson. Discussion opened by Dr. Colin Gurner.

Additional Lectures.

1955.—September 2: By courtesy of Royal Australasian College of Surgeons. Lecture by Dr. D. I. Harris (Professor of Surgery in the University of Toronto). September 26: By courtesy of Royal Australasian College of Physicians. Lecture given by Dr. W. S. C. Copeman. September 6 and October 4: By courtesy of the Australasian Association of Psychiatrists.

1956.—February 7, March 6 and May 1: By courtesy of the Australasian Association of Psychiatrists.

1955.—October 13: By courtesy of Laennec Society (South Australian Branch).

1956.—April 19: By courtesy of Laennec Society (South Australian Branch).

ATTENDANCES AT COUNCIL AND COMMITTEE MEETINGS.

	Council.	National Health Service Sub-Committee.	Scientific Sub-Committee.	Standing Committee on Hospitals.	Library
Bennett, G. L.	10	3	1	1	—
Chinner, M. E.	11	2	1	—	1
Cockburn, M. T.[1]	8	—	—	—	—
Elix, R. H. ..	12	3	—	1	—
Greenlees, R.	12	—	—	—	—
Hamilton, I. A.	10	—	1	—	—
Holmes, R. M.	10	—	—	—	2
Hunter, R. ..	11	2	1	—	—
Jose, I. B. ..	8	1	1	—	—
Jungfer, C. C.	10	3	—	1	—
Mallen, L. R.	10	3	—	—	—
Newland, M. C.[1]	5	—	—	—	—
Oaten, H. R.[2]	4	—	—	—	—
Rieger, C. O. F.	12	3	—	—	—
Verco, P. W. ..	10	—	1	—	—
Wall, F. L. ..	8	3	1	—	—
Meetings held up to June 7, 1956	12	3	1	1	3

[1] Resigned December 10, 1955.

[2] Appointed January 19, 1956.

Dr. W. M. Irwin attended 11 meetings as the Local Representative of THE MEDICAL JOURNAL OF AUSTRALIA.

Dr. J. M. Dwyer attended the three meetings of the Library Committee which were held during the year.

[3] Leave of absence, May 1, 1956.

Membership.

The membership of the Branch is now 851, the number of student associate members being 33.

The deaths of the following members throughout the year are recorded with regret: Dr. R. A. Goode, Dr. D. T. M. Hayes, Dr. J. C. Mayo, Dr. F. H. Beare, Dr. G. H. B. Black, Dr. G. S. Shipway and Dr. W. Gilfillan.

Representation.

Medical Board of South Australia.—Dr. C. O. F. Rieger.

Dental Board of South Australia.—Dr. W. John Close.

Flying Doctor Service of Australia.—Dr. J. M. Dwyer (chairman).

Federal Council of the British Medical Association in Australia.—Dr. L. R. Mallen and Dr. C. O. F. Rieger.

Saint John Ambulance Association.—Dr. H. H. Hurst.

"The Medical Journal of Australia" (local representative).—Dr. W. M. Irwin.

British Medical Hall Company, Limited.—Dr. C. O. F. Rieger, Dr. F. L. Wall and Dr. B. S. Hanson.

Mothers and Babies' Health Association.—Dr. Neville Bickford.

Advisory Council on Health and Medical Services.—Dr. L. R. Mallen.

Nurses' Board of South Australia.—Dr. R. L. Thorold Grant.

University Post-Graduate Committee in Medicine.—Dr. L. R. Mallen, Dr. Ronald Hunter and the President, *ex officio*.

Central Council of the Association, London.—Dr. Myles Formby.

Chiropody Board of South Australia.—Dr. Neville P. Wilson.

Florence Nightingale Memorial Committee.—Dr. Mary Burnell and Dr. Barbara Russell.

World Medical Association.—Dr. L. R. Mallen.

Representatives to Annual Meeting of the Parent Association, Brighton, 1956.—Dr. M. T. Cockburn, Dr. E. P. Cherry and Dr. F. K. Mugford.

Sections Formed for Special Branches of Medical Knowledge.

Section of Anæsthetics.

The annual general meeting was held at the residence of Dr. Mary Burnell on December 12, 1955. The following officers were elected: Chairman, Dr. John Stace; Vice-Chairman, Dr. J. Ferris; Honorary Secretary, Dr. M. J. C. Muirhead; Honorary Treasurer, Dr. E. C. Hallett.

The society is holding its bi-monthly meetings at Newland House this year. Five meetings have been held during the period covered by this report. The Section has 33 members, and the average attendance of 20 has been most gratifying. At these meetings the time occupied by business is kept as short as possible to enable members to hear and discuss the two scientific papers presented at each meeting. All members of the South Australian Branch of the British Medical Association interested in anæsthesia are welcome to attend these meetings.

Dr. Pauline Daniels is the State sub-editor of the *Anæsthetic Newsletter*, a position newly created as a result of a decision made at the annual general meeting of the Australian Society of Anæsthetists held in Melbourne in March this year.

Section of Clinical Medicine: Annual Report for the Year 1955-1956.

Membership.—There are 65 members on the roll, of whom 41 are financial, a decrease of 22 compared with the last year. In this respect it may be mentioned that the Section has now decided to send notices of meetings to those members who have been financial within the last two years. This should result in a considerable saving in financial outlay for postage *et cetera*. It also means that the number of members on the roll officially will be reduced.

Officers.—At the annual general meeting on May 3, 1955, the following officers were elected: President, Dr. F. Ray Hone; Honorary Secretary and Treasurer, Dr. A. Kerr Grant; Committee, Dr. H. G. Rischbieth, Dr. I. M. H. Camens, Dr. H. Lander.

Clinical Meetings.—Four clinical meetings were held throughout the year in May, August and November, 1955, and February, 1956. All meetings took the form of presentation of cases and a wide variety of clinical material was seen. Some recommendations can be made from experience with meetings in the past year.

1. The meeting in February, 1956, took the form of a symposium on the various methods of treatment of thyrotoxicosis. The committee feels that this meeting was a success and recommends a meeting of this type once a year.

2. Three cases of surgical interest were presented during the year, and these resulted in lively discussions and the attendance of a considerable number of surgeons. The Section resolved during the last year to continue to request cases of surgical interest from time to time.

3. The official meeting place has now changed from the Physiotherapy Department to the Chest Clinic, as the Physiotherapy Department is now too small for comfort. The Chest Clinic was used on the February meeting and was found convenient in every way. The committee desires to express its appreciation to Dr. P. Woodruff for his permission to use the Chest Clinic.

4. The average attendance at meetings throughout the year was 35, an increase of five over the average attendance for 1954-1955.

British Medical Association Prize.—The prize for 1955 was won by Mr. J. S. Jose.

The winner of the prize for 1954 was presented to the President of the British Medical Association by the President of the Section at the annual general meeting of the British Medical Association in 1955. This was the first time that the winner of the prize has been officially recognized in this manner.

Section of Ophthalmology.

The following is the annual report of the Section of Ophthalmology, South Australian Branch of the British Medical Association.

Seven meetings were held during the year. Membership was sixteen and the average attendance at meetings was eleven.

The annual general meeting was held on March 8, 1955, and the following officers were elected: Chairman, Dr. Charles Swan; Vice-Chairman, Dr. D. W. Brummitt; Honorary Secretary and Treasurer, Dr. J. H. Slade; Committeeman, Dr. F. J. B. Miller; Auditor, Mr. F. C. W. Dobbie.

In all, 42 cases were shown and discussed at meetings.

In March last year a special meeting was held at the home of Dr. Crompton. At this meeting Mr. Mackie, of Sheffield, delivered three papers on: (i) miscellanea (this included many practical personal ideas of the speaker); (ii) placental implants in *retinitis pigmentosa*; (iii) glaucoma.

On May 10 Mr. T. R. Dinning delivered a paper on "Visual Symptoms in Neurological Disease". It was later suggested to Mr. Dinning that this excellent paper should be published.

At a meeting held on September 13, Dr. Goddard and Dr. Rogers addressed members on their overseas experiences.

At a special meeting held on March 7 last, members were addressed by Mr. Pinkerton, of Honolulu. Mr. Pinkerton read a paper on "Recurrent Pterygia", and showed many slides on pterygia and a film on the Pan-Pacific Congress.

At the remainder of the meetings most of the time was devoted to the examination and discussion of cases.

We record with regret the death during the year of Dr. G. H. B. Black, who was one of our most enthusiastic members and whose sound advice was a great help to all members.

Ear, Nose and Throat Section.

The inaugural meeting of the Section was held on Tuesday, March 8, 1955, following the final meeting of the South Australian Branch of the British Medical Association, Incorporated, Ear, Nose and Throat Section.

The following officers were elected: Chairman, Dr. R. H. von der Borch; Vice-Chairman, Dr. A. S. de B. Cocks; Committeeman, Dr. S. Pearlman; Auditor, Mr. F. W. C. Dobbie; Secretary and Treasurer, Dr. R. N. Reilly.

Three ordinary meetings and one special meeting were held during the year.

On May 17 a meeting was held at the rooms of Dr. R. N. Reilly. One case was shown. Dr. Reilly presented a paper on impressions gained during a trip abroad to England and the Continent. He had on display a series of twelve mounted specimens of carcinoma of the larynx and hypopharynx which had been presented to him by Mr. C. P. Wilson, Director of the Ferens Institute of Pathology at the Middlesex Hospital, and which would go to form a nucleus of a museum for the Oto-Laryngological Society of Australia. The specimens would be housed temporarily in the Pathological Museum, University of Adelaide, through the kindness of Professor J. S. Robertson.

At Dr. von der Borch's rooms on July 19 five cases were shown. Dr. Matison presented a paper on his experiences during a twelve-month trip to the United States of America, England and the Continent, during which he had spent a good deal of time following up present practices in endoscopy.

On August 11 a special general meeting was held to discuss the formation by the State Government of a Deafness Guidance Clinic. As a result the Council of the South Australian Branch of the British Medical Association, Incorporated, was asked for particulars from the Minister of Health.

At the annual general meeting of the Oto-Laryngological Society of Australia held in Sydney later in August, Dr. R. M. Glynn was elected President, Dr. A. S. de B. Cocks Vice-President, and Dr. R. N. Reilly Secretary-Treasurer. This was the first occasion on which the Executive of the Australian society had been formed of members outside of the eastern States.

The State Section held its final meeting for the year on October 25. Dr. R. F. West presented an interesting and instructive paper on "The New Vascular-Acting Drugs and Their Relation to Oto-Rhino-Laryngology", approaching his subject from both the pharmacological and the clinical points of view.

Affiliated Local Associations of Members.

Northern Medical Association.

There were four meetings held during the year.

The first was held at Gladstone on Sunday, August 7. Ten members were present. There were two speakers: Mr. K. Brown spoke on chest surgery in relation to general practice and Dr. R. Plummer spoke on the management of epistaxis and sinusitis.

The second meeting was held at Yacka on Sunday, November 6, 1955. Fourteen members attended. There were two speakers: Dr. J. S. Covernton spoke on cardiac failure and Dr. G. T. Gibson on "The Prolonged Labour".

The third meeting was an all-day one held at Clare on Sunday, February 4, 1956. There were four speakers. Ten members attended. "Vomiting in Childhood", Dr. E. B. Sims; "Deafness, Otitis Media and Adenoids", Dr. Peter Jay; "Hypertension: Medical Aspects", Dr. Mark Bonnin; "Hypertension: Obstetrical Aspects", Dr. L. O. S. Poidevin.

The final meeting was held at Port Pirie on Sunday, April 8, 1956. There were two speakers: Dr. J. M. McPhie, "Acute Cardio-Vascular Emergencies", and Mr. J. R. Magarey, "Acute Abdominal Emergencies". Twelve members attended.

Arrangements for lectures and lecturers have now been taken over by the Post-Graduate Committee in Medicine, and this seems to be working very satisfactorily. Comprehensive tickets costing £10 10s. a year are available to members entitling them to attend all local meetings and also all interstate and overseas lectures, refresher week, medical ward rounds, obstetrical tutorials and any twelve selected lectures for a higher degree course.

South-Eastern Medical Association.

Four meetings were held during the year.

On July 23, 1955, at Millicent, a clinical meeting was held, followed by a dinner, after which Dr. John Game gave an address on "Paroxysmal Disorders of the Nervous System".

On October 15, 1955, at Mount Gambier, a clinical meeting was held which was again followed by a dinner, after which Mr. Eric Price gave an address on "Congenital Deformities of the Lower Limb and Their Management".

On February 11, 1956, a clinical meeting was held at Naracoorte, followed by a dinner, after which Dr. D. G. McKay spoke on "Recent Advances in Pædiatric Surgery". This meeting was also attended by Dr. Lindsay Male, who explained the workings of the Rehabilitation Branch of the Department of Social Services.

On April 21, 1956, the annual general meeting was held at Mount Gambier, when the following officers were elected for the ensuing year: President, Dr. R. H. Jarvis; Vice-President, Dr. H. R. Oaten; Secretary and Treasurer, Dr. J. E. Dunn; Committee, Dr. J. H. Nicholls and Dr. I. G. Campbell.

A clinical meeting was held, and after dinner Dr. A. J. M. Sinclair gave an address on "The Diagnosis and Management of the Restless Patient".

In addition, a refresher course was held on July 9, 1955, when the visiting lecturers were Professor R. Hare, Dr. K. V. Sanderson and Dr. J. S. Covernton.

There are 28 financial members, and the average attendance at the meetings has been 15 members.

Upper Murray Medical Association.

Membership.—There are eleven members of the Association.

Local Meetings.—The first meeting was held at Barmera on July 23, 1955, when six members attended. Medical cases were presented for discussion.

The annual general meeting was held at Berri on March 10, 1956, when the following officers were elected: President, Dr. J. G. Wilson; Immediate Past President, Dr. R. T. Auricht; Secretary-Treasurer, Dr. T. D. Manthorpe. Dr. Kohler gave a talk on overseas post-graduate work.

Post-Graduate Week-Ends.—The following post-graduate week-ends were held during the past year:

1955: June 25-26 at Berri. Eight members attended. The visiting lecturers were: Dr. Schneider, "Red Eye"; Dr. Dinning, "Head Injuries"; Dr. Gordon, "Medical Neurology".

1955: September 10 at Barmera. Ten members attended, including a Mildura visitor. Dr. Peter Verco, "X Ray in General Practice"; Dr. Neville Wilson, "Backache"; Dr. Ivan Magarey, "Respiratory Disease in Children".

1955: November 19 at Renmark. Twelve members attended, including a Mildura visitor. Dr. Kerr Grant, "Dyspepsias"; Dr. Heighway, "Gynæcological Emergencies"; Dr. Donald, "Common Skin Diseases".

1956: February 4 at Loxton. Nine members attended. Dr. Hakendorf, "Common Skin Diseases"; Dr. Burston, "Thyroid Disease"; Dr. H. E. Pellew, "Vaginal Discharge".

1956: April 21 at Berri. Eight members attended. Dr. H. Gilmore, "Common Heart Diseases"; Dr. G. T. Gibson, "Pregnancy Toxæmia"; Dr. R. N. Reilly, "Otitis Media".

Yorke Peninsula Medical Association.

The inaugural meeting of the association was held at Wallaroo Hospital on February 12, 1956. Eleven members were present and apologies received from three others. A set of rules was adopted, and it was decided to seek affiliation with the South Australian Branch of the British Medical Association. The following office bearers were elected for the ensuing year: President, Dr. F. L. Thyer; Secretary-Treasurer, Dr. A. J. de Bruin. The main business of the day was a post-graduate refresher course arranged in conjunction with the Post-Graduate Committee in Medicine at the University of Adelaide. The speakers were Dr. E. F. Gartrell and Dr. C. O. F. Rieger. Post-graduate meetings have also been arranged for July and October, 1956.

Publication of Doctors' Names in "Birth Column" Announcements.

During the year further efforts were made by the Council to enlist the support of members of the profession themselves and of hospitals with a view to suppressing publication of the names of attending doctors in "birth column" notices. Only on comparatively few occasions do the names of doctors now appear.

Prescribing of Antibiotics.

In January, 1956, the Dean of the Faculty of Medicine convened a meeting of teachers and others (35 in all) to consider the problem of the use of antibiotics. Subsequently each member of the British Medical Association in South Australia received a circular stressing the fact that incautious prescribing of antibiotics was creating a grave medical problem by developing more and more resistant organisms *et cetera*, and exhorting all to exercise the greatest caution in prescribing and to resist pressure, either from the public or those financially interested, to prescribe antibiotics unnecessarily.

Medical Benefits Scheme.

During the year the Council was again requested to ask members to render to their patients properly itemized accounts. In supporting this request the Council emphasized that proper itemization of accounts on the first time of rendering would save the clerical time of the doctors' staff and would obviate delay for the patient in receiving a refund from the benefits organization. The matter was brought up by the largest voluntary insurance fund operating in this State. The organized medical profession has long supported the principle of voluntary insurance to assist patients to meet the cost of medical care. It is in the interests of all that voluntary insurance schemes retain the support of the public.

Heroin.

Early in March the Council received a communication from the Federal Council of the British Medical Association in Australia, requesting the views of members on the banning of heroin. This ban was previously imposed by the Commonwealth in 1953, after asking the Federal Council to give careful consideration to the proposed prohibition.

In April a *questionnaire* was dispatched to members of the Branch asking them to indicate their views on this important matter. The Federal Council was later advised that the poll overwhelmingly indicated that members favoured the lifting of the present ban imposed on the importation of heroin into Australia.

Medical Certification.

Several complaints of alleged wrongful medical certification received the attention of the Branch Council during the year. In one particular case the member gave a certificate for the maximum period he anticipated that the illness would last, rather than adopting the usual practice of giving a certificate for a shorter period and extending this should

BRITISH MEDICAL ASSOCIATION (SOUTH AUSTRALIAN BRANCH).

Balance Sheet as at December 31, 1955.

LIABILITIES.	£ s. d.	£ s. d.
Sundry Creditors		230 4 7
General Fund:		
Balance, January 1, 1955	7,997 4 4	
Add Net Surplus for year	194 7 9	
Add Transfer from Building Fund	561 6 0	
	8,752 18 1	
		£8,983 2 8

ASSETS.	£ s. d.	£ s. d.
Fixed Assets at cost, *less* Depreciation:		
Furniture and Plant	1,976 19 0	
Delineascope and Projector	5 18 5	
Lister Medals	2 0 0	
		1,984 17 5
Current Assets:		
British Medical Hall Company, Limited, Shares at Cost—		
192 Preference, 134 Ordinary	3,297 10 0	
Australasian Medical Publishing Company, Limited—		
5% Debentures	30 0 0	
3½% Debentures	2,300 0 0	
Advance not yet converted	8 13 1	
		5,636 3 1
Stocks on hand	369 4 3	
Sundry Debtors	603 3 10	
		972 8 1
Cash at Bankers—		
National Bank	255 17 3	
Commonwealth Savings	10 2 7	
Savings Bank of South Australia	100 5 7	
Savings Bank Library	1 18 0	
Cash in Hand	21 10 8	
		389 14 1
		£8,983 2 8

Special Funds and Investments.

	£ s. d.	£ s. d.
Listerian Oration Fund:		
Balance, January 1, 1955	437 7 6	
Add Net Income for year	22 2 1	
	459 9 7	
Less Oration Expenses	50 6 0	
		409 3 7
Frank S. Hone Memorial Fund:		
Balance, January 1, 1955	688 12 10	
Add Interest	32 5 11	
	720 18 9	
Less Awards	31 10 0	
		689 8 9
Trent Champion de Crespigny Memorial Fund:		
Balance, January 1, 1955	273 14 4	
Add Interest	6 16 6	
		280 10 10
British Medical Hall Building Fund:		
Balance, January 1, 1955	1,005 2 11	
Add Interest, Donations *et cetera*	577 16 8	
	1,582 19 7	
Less Transfer General Fund	561 6 0	
		1,021 13 7
		£2,400 16 9

	£ s. d.	£ s. d.
Listerian Oration Investments:		
Electricity Trust Debentures	310 0 0	
Savings Bank of South Australia	99 3 7	
		409 3 7
Frank S. Hone Memorial Fund Investments:		
Commonwealth Bonds, 3¼%, 1959	50 0 0	
South Australian Gas Company Debenture	300 0 0	
E.T.S.A. Debenture	250 0 0	
Savings Bank of South Australia	89 8 9	
		689 8 9
Trent Champion de Crespigny Memorial Fund:		
Savings Bank of South Australia		280 10 10
British Medical Hall Building Fund:		
Comonwealth Bonds, 3¼%, 1960	200 0 0	
Savings Bank of South Australia	501 8 7	
Commonwealth Savings Bank General Account	320 5 0	
		1,021 13 7
		£2,400 16 9

F. C. W. DOBBIE, Secretary. F. L. WALL, Honorary Treasurer.

We hereby report that we have examined the Books and Accounts of the South Australian Branch of the British Medical Association, Incorporated, for the year ended December 31, 1955. In our opinion, the above Balance Sheet, together with the Statements of the Special Funds, are properly drawn up so as to exhibit a true and correct view of the affairs of the Branch, as at the above date according to the best of our information, the explanations given to us, and as shown by the Books produced.

Adelaide,
May 8, 1956.

ANNELLS, TILLEY, HUNWICK AND COMPANY,
Chartered Accountants (Aust.).

this be necessary. The attention of the member was directed to this.

Certificates written carelessly in connexion with claims for sick pay through absence from work may provoke legal action against the writer.

It is unnecessary to stress the need for exercising the utmost care and responsibility when issuing a medical certificate.

Immunization Against Tetanus.

Early in November the Branch Council appointed a special subcommittee consisting of Dr. M. T. Cockburn (chairman), Dr. R. Greenlees, Dr. A. H. Lendon, Dr. I. A. Hamilton and Dr. Ronald Hunter, with Dr. J. E. McCartney coopted, to investigate the current practice of giving an injection of antitetanic serum to patients admitted to hospital as a result of traffic accidents. This matter was first brought

BRITISH MEDICAL ASSOCIATION (SOUTH AUSTRALIAN BRANCH).

Income and Expenditure Account for the Year Ended December 31, 1955.

1954. £		£	s.	d.
2,053	To Staff Salaries and Superannuation	2,349	19	8
261	„ Postages	349	7	0
542	„ Stationery and Printing	454	1	10
30	„ Telephone and Telegrams	75	16	6
189	„ Rent	169	15	0
15	„ Legal Expenses	28	17	6
15	„ Audit Fee	15	15	0
274	„ General Expenses	413	5	8
82	„ Entertainment	122	18	2
106	„ Depreciation	219	2	7
	„ Net Surplus for year	194	7	9
		£4,393	6	8

1954. £		£	s.	d.	£	s.	d.
6,902	By Gross Subscriptions				7,410	19	3
5,391	City	5,578	8	11			
1,428	Country	1,781	6	10			
83	Students	51	3	6			
2,826	*Less* Deductions:				3,138	2	10
1,170	British Medical Association, London	1,365	3	10			
339	THE MEDICAL JOURNAL OF AUSTRALIA	391	7	6			
867	Federal Council	831	11	6			
450	Library Grant	550	0	0			
4,075	By Net Subscriptions				4,272	16	5
86	„ Interest				105	0	3
25	„ N.H.S. Prescription Pads				15	10	0
					£4,393	6	8

under notice by Professor J. B. Cleland in the course of his remarks at the October scientific meeting, the subject of which was "The Prevention of Motor Accidents and the Medical Profession".

Two meetings of the subcommittee were held and several recommendations made. These were then referred to the National Health and Medical Research Council for further comment and advice.

Proposed Establishment of "Chamil" Clinic by South Australian Chamber of Manufactures Insurance, Limited.

After two conferences had taken place with the local manager of the company and its chief medical adviser in this State, the Council decided to refer the matter of the proposed establishment of this clinic for the treatment of workers' compensation injuries to an extraordinary general meeting of members.

At a very well-attended meeting which was held on March 22, 1956, the following resolution was adopted.

> This meeting expresses its opposition to the establishment of an insurance company controlled medical clinic.

The Federal Council was later informed of this.

The question of ethics to be followed by medical practitioners in treating workers' compensation cases in medically controlled clinics has been referred to the Ethics Committee for consideration and report.

Dr. A. Talbot Rogers.

Dr. Talbot Rogers, who is a member of the Central Council and chairman of the General Medical Services Committee of the British Medical Association, visited Australia in August, 1955, when he acted as the representative of the Parent Association at the Ninth Session of the Australasian Medical Congress. He later visited South Australia (where he spent his early years), and during the course of this he was entertained by the Council at "Newland House".

Visit of New Health Minister.

Dr. Donald Cameron (who succeeded Sir Earle Page as Federal Minister for Health) was given a buffet dinner by the President of the Branch on January 24, 1956, on the occasion of his first official visit to South Australia.

Dr. Cameron, accompanied by the Director-General of Health (Dr. Metcalfe), later attended a meeting in the Council chamber at "Newland House".

The World Medical Association.

The Secretary-General of the World Medical Association (Dr. Louis H. Bauer), who came to Australia to deliver the Henry Simpson Newland Oration at the Ninth Session of the Australasian Medical Congress, visited Adelaide on Saturday, August 13, 1955.

Dr. Bauer was entertained by the Council at dinner and afterwards addressed members on "International Medicine, with Special Reference to the Aims and Activities of the World Medical Association".

Dr. L. R. Mallen attended Council meetings of the World Medical Association held at Vienna and Cologne in October, 1955, and April, 1956, respectively.

Federal Council of the British Medical Association in Australia.

The Federal Council met in Sydney on August 17 to 24, 1955, and in Hobart on February 27 to March 1, 1956. The Branch was represented at these meetings by Dr. L. R. Mallen and Dr. C. O. F. Rieger.

Following the death of the President of the Federal Council (Sir Archibald Collins), Dr. L. R. Mallen was elevated to the office of Chairman of the Federal Pharmaceutical Benefits Advisory Committee.

Sir Archibald Collins was elected President of the Federal Council in 1951, and held this office until his death on June 24, 1955. The medical profession in Australia will ever appreciate the qualities of leadership which he displayed in negotiations between the Federal Council and the Commonwealth Government.

The sympathy of the Branch was extended to Lady Collins and family.

Frank S. Hone Memorial Prize.

The prize awarded on the results of the 1955 examinations for the degrees of Bachelor of Medicine and Bachelor of Surgery was awarded to Kenneth Duncan Fitch.

Honours and Awards.

The congratulations of the Council are extended to Dr. L. J. Pellew and Dr. J. M. Dwyer, on whom Her Majesty the Queen has been pleased to bestow the awards of C.B.E. and O.B.E. respectively, and to Dr. J. M. Pedler, who has been admitted in the grade of Serving Brother in the Venerable Order of St. John of Jerusalem.

The above represents but a brief summary of some of the matters which have engaged the attention of the Branch Council throughout the period under review. The past year has been a very active one, and I would like to thank the Immediate Past President (Mr. I. B. Jose) for deputizing for me in November-December, 1955, during the period of my illness.

(Signed) G. L. BENNETT,
President.

FINANCIAL STATEMENT.

The financial statement, published herewith, was adopted on the motion of Dr. F. L. Wall, the Honorary Treasurer, seconded by Dr. C. O. F. Rieger. In seconding the motion, Dr. Rieger expressed regret that Dr. Wall was presenting the financial statement for the last time, as he had found it necessary, owing to ill health, to relinquish the office of honorary treasurer which he had held for seven years. The financial statement is published herewith.

ELECTION OF OFFICE BEARERS.

In the absence of other nominations, Dr. G. L. Bennett declared Dr. M. E. Chinner elected to the office of President for the ensuing twelve months and invested him with the badge of office. Dr. Chinner thanked the members for his election.

Dr. Chinner announced the election of office bearers for the ensuing year.

Vice-President: Dr. Peter Verco.

Honorary Treasurer: Dr. B. S. Hanson.

Honorary Medical Secretary: Dr. Ronald Hunter.

Members of Council: Group A—Dr. J. R. Barbour, Dr. G. T. Gibson, Dr. J. W. Sangster. Group B—Dr. R. N. Holmes, Dr. H. R. Oaten.

On the motion of Dr. F. L. Wall, seconded by Dr. C. O. F. Rieger, Messrs. Annells, Tilley, Hunwick and Company were appointed auditors for the ensuing year.

Prize for Clinical Medicine.

The chairman of the Section of Clinical Medicine, Dr. John Hayward, presented to Dr. Bennett the winner of the Section of Clinical Medicine Prize, Mr. J. S. Jose. Dr. Bennett congratulated Mr. Jose on having won the prize and handed him a certificate of the award.

Retiring Members of Council.

A vote of thanks to the retiring members of Council—Dr. Ivan B. Jose, Dr. F. L. Wall, Dr. R. Greenlees and Dr. R. H. Elix—was passed on the motion of Dr. A. Britten Jones, seconded by Dr. Ian Hamilton.

Retiring President's Address.

Dr. G. L. Bennett read his retiring president's address entitled "Some Thoughts on General Practice, Yesterday, Today and Tomorrow" (see page 329).

A vote of thanks to Dr. Bennett for his address was passed on the motion of Dr. L. R. Mallen, seconded by Dr. C. C. Jungfer.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PREVALENCE OF VENEREAL DISEASE IN SYDNEY COVE.[1]

[Governor Arthur Phillip to Lieutenant P. G. King, Commandant at Norfolk Island, February, 1789.]

ALTHOUGH every precaution is taken to prevent any person being sent who may be infected with the venereal complaint, it is possible that the Surgeon may be deceived therefore Mr. Jamison will make a Strict Enquiry and if the disorder ever apears on the Island you will take every step to eradicate it effectually that you would do if it was the Plague. It is a more severe Scourge and it has gained such a footing in the Settlement that I now doubt if it will ever be done away with. The Precautions I ordered to be taken in England have been rendered ineffectual by the infamous conduct of some of the Magistrates, in sending on board the transports those who have been turned out of the Hospitals as incurable.

Correspondence.

THE CENTENARY OF SIR ALEXANDER MacCORMICK'S BIRTH.

SIR: It is probable that the fact that July 31, 1956, marks the centenary of the birth of that famous surgeon, the late Sir Alexander MacCormick, is known to only a small quota of his admirers—that is, apart from his relatives and personal friends.

It is now over fifty years since the present writer was house surgeon to Sir Alexander at one of the large Sydney hospitals. He has always admired the consummate art of the surgeon, but *en passant* he may say that he not only admired but envied the surgeon his indefatigable energy. Mr. MacCormick would say: "I shall be here on Tuesday morning at 7 to operate." "Very good, sir." But he would arrive in the hospital and be there before 6.30 a.m., and they would ring from the theatre to say he was waiting for me, and often this particular house surgeon went without breakfast, not to keep the great man waiting. The surgeon himself had already travelled from Port Hacking (some twenty miles away). At 2 p.m. he returned, to operate again until 6 p.m.

FIGURE I.
Le Val, Saint Brelade, Jersey.

In 1950 I had the opportunity of taking a third trip to the United Kingdom—this time by air—and I determined to visit Jersey by air—principally to pay my respects to Lady MacCormick, and with her permission (which was readily granted) to take photographs of Sir Alexander's grave and her home—"Le Val", Brelade, Jersey.

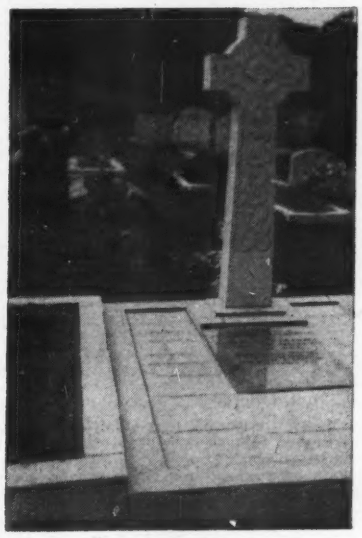


FIGURE II.
Sir Alexander MacCormick's grave, Saint Brelade, Jersey.

Lady MacCormick's home is a very fine and very modern home, and has very extensive grounds—it is quite a long distance from the lodge at the entrance down to the house. The house is situated in a valley, and there were two gardeners working—the gardens were a blaze of colour.

Lady MacCormick received me in a morning room which opened on to a flagged terrace where her husband used to love to sit and read; he did not have a long illness, but passed peacefully away during the night. At the bottom of the grounds is a bay (Brelade), and there is a martello tower; many were built along the southern coast of England during the Napoleonic wars to prevent invasion by Napoleon.

[1] From the original in the Mitchell Library, Sydney.

Sir Alexander's grave is in the Saint Brelade churchyard. On it is a white marble cross, and set in the white marble tiles on the grave is the following inscription:

TO THE
BELOVED MEMORY OF
ALEXANDER MACCORMICK
K.C.M.G.
M.D. M.R.C.S. HON. F.R.C.S. ENG. EDIN.
COL. R.A.M.C. 1899-1900 (1914-1918)
OF SYDNEY, N.S.W.
BORN TAYNISH, ARGYLL
31ST JULY 1856
DIED LE VAL, ST. BRELADE
25TH OCTOBER 1947

Yours, etc.,

P. E. WALTON SMITH.

"Mylora",
30 Bondi Road,
Bondi Junction,
New South Wales.
July 16, 1956.

THE MEDICAL BENEFITS FUND OF AUSTRALIA AND SERVICES RENDERED IN PUBLIC WARDS OF HOSPITALS.

SIR: With your permission and after mentioning my intention to Dr. A. J. Metcalfe, Director-General of Health, in advance of publication I desire to refer to the first and last paragraphs of his letter of July 27, 1956, published in THE MEDICAL JOURNAL OF AUSTRALIA, August 25, 1956.

In his first paragraph he connects the question of benefit payments with services rendered in public wards. In giving the relevant facts in my letter of July 14, 1956, I referred to services rendered to patients classified as public. It is important to bear this distinction in mind, as there is no difficulty whatever in the payment of benefits to those classified intermediate or private.

In the third paragraph Dr. Metcalfe states: "The organizations, however, are not prohibited from paying fund benefits. They are free to determine their own policy in such matters." I have pointed out to the Director-General that that cannot be correct in the light of the department's circular NM13 which I now have no alternative but to publish.

Commonwealth of Australia,
Department of Health,
39 York Street,
Sydney.
21st December, 1954.

The Public Officer,
All Registered Medical Benefit Organizations in New South Wales.

Dear Sir,

Medical Benefits Scheme: Fees Charged by Medical Practitioners to Public Ward Patients.

In view of the provisions of Section 36 of the New South Wales *Public Hospitals Act*, organizations registered under the *National Health Act*, 1953, must not condone an offence against State law by paying benefits in respect of medical expenses which are not lawful.

All registered medical benefit organizations in this State are therefore required to include in their claim form the question: "If you were a patient in a public hospital, were you classified as an intermediate or private patient?" If your claim form does not already include the above or similar question, would you please arrange for it to be amended accordingly as early as possible?

Yours faithfully,

C. R. WIBURD,
Deputy Director of Health, N.S.W.

Mr. C. J. Eade, Honorary Secretary of the Association of Registered Hospital and Medical Benefit Organizations in New South Wales, informs me that all funds in New South Wales interpret this instruction as does the Medical Benefits Fund of Australia as prohibiting the payment of any benefit in respect of patients classified public. It is difficult to see what other meaning could be given the words of the instruction. In any case payment of a fund benefit to such patients who pay no fee at all would be an infringement of the 90% rule and would render funds liable to loss of registration.

Yours, etc.,

H. R. R. GRIEVE,
Chairman, Medical Benefits Fund of Australia.

113 Homer Street,
Undercliffe,
August 27, 1956.

ENCEPHALITIS LETHARGICA.

SIR: In my article "Changing of Disease Patterns, Disappearance of Disease, New Diseases" (M. J. AUSTRALIA, January 21, 1956) I wrote that Burnet described the spontaneous disappearance of three virus diseases in 1953. One of them is von Economo's *encephalitis lethargica*. He wrote: "No certain case of this disease has been reported since 1930." It was, therefore, of special interest for me to read an article in the *British Medical Journal* (May 19, 1956) of M. L. E. Espir and J. M. K. Spalding, in which the authors report three cases of acute encephalitis and "have no doubt that these three cases should be regarded as examples of von Economo's encephalitis lethargica". I myself find the description of the disease of these three patients and the argumentation of the authors very convincing (there exists no specific test which would confirm the diagnosis). So it seems that *encephalitis lethargica* did not disappear as completely as seemed to be the case.

Yours, etc.,

CHARLES ENGEL.

201 Macquarie Street,
Sydney,
August 6, 1956.

HEAD INJURIES.

SIR: The leading article in THE MEDICAL JOURNAL OF AUSTRALIA of August 4 rightly draws attention to the tragic loss of life occurring nowadays from accidents especially due to head injuries. In it, certain measures are recommended to improve the diagnosis and treatment of the head injured, but no mention is made of the decided advantages that have resulted overseas in such cases, from their handling by an accident service which integrates, and makes readily available, the modern advances in all fields of surgery.

Australia is well provided with neurosurgeons, but under a system where the majority of head injuries are admitted to general surgical wards under general surgical routines, their specialized knowledge is often unnecessarily delayed or not called upon in practice.

Hence it is advocated that a properly coordinated accident service be developed in the leading teaching hospitals in Australia to treat cases, and teach the principles of traumatic work along the lines advantageously employed in the United Kingdom over the past fifteen years.

Yours, etc.,

GEOFFREY VANDERFIELD.

The Royal Prince Alfred Hospital Medical Centre,
100 Carillon Avenue,
Newtown.
August 6, 1956.

Obituary.

KEVIN BYRNE.

DR. KEVIN BYRNE, whose death was announced in this journal some weeks ago, represented a type of practitioner much to be desired. He made his patients his first consideration, he practised the ethics of his profession, and he tried to keep himself abreast of the newer knowledge in medicine.

Kevin Byrne was born at Wallsend, near Newcastle, in 1891. He studied medicine at the University of Sydney and graduated M.B., Ch.M. at the beginning of 1915. After serving as resident medical officer at Saint Vincent's Hospital, Sydney, he commenced practice at Lakemba. How-

ever, he wanted to join the Navy and go on active service. He loved the sea and everything connected with it. He had to wait some time for his call to join up, and when it did come he just walked out of his house leaving a notice stating that he was off to the war and would see his patients when he came back. He was given the rank of surgeon lieutenant and served with the sea-going division. Having returned from active service, he went back to Lakemba, the site of his first practice, where he soon built up an extensive practice. He served for a period as honorary medical officer at Saint Joseph's Hospital, Auburn, and for a longer period at the Canterbury District Memorial Hospital. He was a loyal member of the British Medical Association, and being

widely read in medical and general literature, was helpful to this journal on many occasions in the 1930's. He was intolerant of regimentation and showed his resentment in no uncertain way in the early attempts of the Federal Government to bend the medical profession to its will. Unfortunately he did not enjoy good health, and in 1951 he was compelled to retire from practice. In his long and trying illness he showed the courage that one would expect of a man of his convictions and calibre. Much sympathy has been expressed with his widow and children.

DR. R. W. MCCREDIE writes: Kevin Byrne graduated from the University of Sydney in 1915 and saw active service during the first World War as a surgeon lieutenant in the Royal Australian Navy. After the cessation of hostilities, he returned to his practice at Lakemba, where he remained for thirty-four years. His life was a full one, with a diversity of interests, and his frequent periods of ill health were accepted with fortitude and then virtually ignored, only his last lengthy illness necessitating the abandonment of his activities.

A forceful personality, he lived his convictions with a strength of purpose leavened by a keen, at times a devastating, sense of humour. To his patients he was the true family doctor—guide, philosopher and friend. His character imprinted itself on the district; his patients held him in deep respect, but above all loved him and remember him for his charity, exercised especially during the depression years. He is survived by his wife, two sons and two daughters.

HAROLD RUSSELL LOVE.

WE have received from Dr. K. B. Fraser the following appreciation of the late Dr. Harold Russell Love.

Harold Russell Love, the son of Harold William Love, was born on August 31, 1903, at Mareeba in North Queensland, his father being at that time the manager of the Bank of North Queensland there. He commenced his secondary education at the Brisbane Grammar School, where he won the Lilley Silver Medal in the Fifth Form. Later he transferred to Scotch College, Melbourne, and won the Alex Morrison Memorial Gold Medal and an exhibition to the University of Melbourne. Graduating M.B., B.S. in 1926, he became a resident medical officer at the Royal Melbourne Hospital. Returning to Queensland in 1927, he commenced practice as an assistant to his uncle, Dr. Wilton Love, then a leading practitioner in Brisbane. Some years later he went into general practice in Paddington, Brisbane, and it was from here that he enlisted in the Australian Imperial Force on the outbreak of war in 1939. He was posted as a major to the 2/3rd Field Ambulance, and left for overseas in May, 1940, sailing on the *Queen Mary* in a convoy that was diverted to England. He soon proved himself a capable ambulance officer and quickly endeared himself to his men, but he really came into his own in the crowded and convivial atmosphere of the old-fashioned inns of Salisbury Plain, where his knowledge of food and wine and his ready wit soon became a byword. Not long after moving from England to the Middle East in March, 1941, he found himself in Tobruk, where he served throughout the siege.

Placed in charge of a large ward of physically exhausted and badly shaken men, his staunch outlook and vigorous personality restored their confidence and morale to such an extent that the great majority of them were soon back with their units.

He had always had a leaning towards medicine, and his experiences in Tobruk, which were recorded in THE MEDICAL JOURNAL OF AUSTRALIA and received wide recognition, set him on the path which he was to follow with such distinction.

After some time with 2/2 Australian General Hospital at Kantara as a physician, he found himself back in Australia in March, 1942. Quickly gaining his M.R.A.C.P., he was promoted lieutenant-colonel and posted as Officer Commanding Medical Division 115 (Heidelberg) Military Hospital. From then until the end of the war he was officer commanding medical division of two other large military hospitals, eventually being discharged in January, 1946, to take up practice as a physician in Brisbane, where he was appointed a junior visiting physician at the Brisbane General Hospital.

In 1947 he became a senior visiting physician, and was appointed also chest physician at the South Brisbane Auxiliary Hospital and consulting physician to the Repatriation Commission. In 1950 he became a Fellow of The Royal Australasian College of Physicians.

Soon after recommencing practice at the end of the war, he was elected to the Council of the Queensland Branch of the British Medical Association; after a term as secretary in 1947 he became State President in 1950, and his term of office as President completed, he remained on the Council until his death.

Harold Love had at times what almost amounted to a phase of lethargy; but this was soon over, and normally his very considerable output of effective work was achieved with no apparent effort or hurry. His kindness and thoughtfulness for all his patients were proverbial, but I think he derived the greatest pleasure of all from the psychosomatic aspects of his work.

His activities apart from his profession were many and varied. Music, painting and literature all held his interest; he was a voracious reader and painted quite well himself. He was really seen to best advantage in his own home, surrounded by his family. Some years before his death he bought a big, rambling house with large grounds at Toowong, and I never saw him happier than when he and his wife were entertaining their friends in this congenial atmosphere. He had the happy knack of making friends with all sorts and conditions of people; in fact, people interested him perhaps more than anything else. He loved good company and delighted in the almost forgotten art of conversation. Indeed, I often had the feeling that he belonged rightly to a past generation, and it was easy to imagine him reclining at his ease, taking his snuff and discoursing on the topics of the day.

One would have expected him to be a facile and brilliant speaker, for he was widely read and had an intelligence far above the average; in debate, however, his thoughts sometimes flowed too quickly for his tongue, and his clever penetrating mind showed to best advantage in his writings.

Approaching the peak of his career and with a great future before him in his profession, ill health beset his path before he was fifty. He continued to work, however, as hard as ever, until he died, quite suddenly, on April 17, 1956, while he was quietly preparing a paper, and with a book which he was writing still only half-finished.

With all his other friends in many walks of life I feel sad indeed at his untimely end. Some lines from Tennyson's "In Memoriam" seem to me to express what those of us who knew him so well and loved his robust humanity must all be feeling:

I leave thy praises unexpress'd
In verse that brings myself relief,
And by the measure of my grief
I leave thy greatness to be guess'd.

For can I doubt, who knew thee keen
In intellect, with force and skill
To strive, to fashion, to fulfil—
I doubt not what thou wouldst have been.

Our deepest sympathy is extended to all the members of his family.

JAMES WILLIAM HEASLOP.

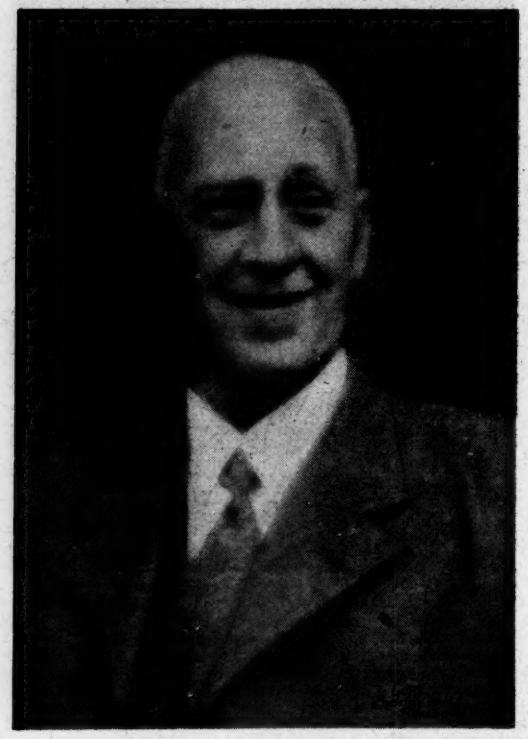
WE have received from Dr. B. B. Barrack the following appreciation of the late Dr. James William Heaslop.

James William Heaslop, who died on June 2, 1956, known to most of his friends as "Wally", was one of the first medical men to specialize in dermatology in Queensland. He was the eldest son of the late Mr. and Mrs. James Heaslop, pioneers of South Brisbane, and was born on February 1, 1879. He was educated at Toowoomba Grammar School, and from there he went to the University of Sydney, where he was in residence at Saint Andrew's College. He graduated M.B., Ch.M. in 1908. At the university he was keenly interested in cricket, football and rowing, and he represented his university in the latter sport (I think he stroked the eight).

After graduation he joined the staff of the Brisbane General Hospital, and later went into practice at Kingaroy, where he remained until 1919. It was a tribute to his ability, and the esteem in which he was held by the people of that district, that they sought his help and advice right up to the time of his death some forty years later. During the pneumonic influenza epidemic he acted as port doctor in Brisbane.

After leaving Kingaroy he did post-graduate work in London, where he was associated with Dr. Dore, and in Edinburgh under Sir Norman Walker, to whom he was appointed assistant. He did further work at the Vanderbilt Clinic in New York, when it was the most famous skin clinic in North America. He returned to Brisbane in 1922 and started practice in dermatology and radiology in Wickham Terrace, and continued to practise there till a few weeks before he died. He served on the honorary staffs of the Brisbane General Hospital and the Brisbane Children's Hospital, and in later years, although not attached to any hospital, he never failed to lend a willing hand to help when shortage of dermatologists created difficulties, particularly during the second World War.

Wally Heaslop was liked by all; he embodied in his specialist practice all the kindliness of the old type of family doctor, and that, combined with a sound knowledge of his



work, leaves a gap that will be difficult to fill. He retained in his later years a great love for the country, and he was never happier than when he spent his holidays with his wife and his boys touring the countryside and camping out—it was extraordinary how quickly he could select his site, pitch his tent and make things comfortable. He enjoyed the simple things of life, and it was the country and the life near to nature that gave him his greatest pleasure. He was married twice. His first wife predeceased him, as did his only son Hardie by this marriage; this was a severe blow, but in spite of it he carried on. He married again in 1944, Miss Gwen Isaacs, and by his second marriage he leaves two young sons. Our deepest sympathy goes out to them and their mother. They will have a very splendid memory of one who had no enemies, who did a great job and who was loved by all.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 39, of July 19, 1956, and Number 40, of July 26, 1956.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Fixing Rates of Pay.—Surgeon Lieutenant (for Short Service) Joseph Brian Shiels is paid the rates of pay and allowances prescribed for Surgeon Lieutenant-Commander, whilst acting in that rank, dated 21st May, 1956.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Alan Morton Cuthbertson is appointed Surgeon Lieutenant with seniority in rank of 20th December, 1955, dated 20th March, 1956.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

The Short Service Commission granted to 3/40054 Major (provisionally) J. H. Cater is extended until 25th June, 1957.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/62657 Captain R. A. Rimington is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command), 1st December, 1955.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of the following officer is terminated: Captain F3/1019 P. B. Blackall, 24th December, 1955. 3/82441 Major D. C. Cowling ceases to be seconded whilst in the United Kingdom, 16th April, 1956. 3/50235 Major B. T. Keon-Cohen is appointed from the Reserve of Officers, is appointed Consultant Orthopædist, Army Headquarters, and to be Lieutenant-Colonel (Temporary Colonel), 12th June, 1956. 3/101823 Captain (provisionally) K. H. McLean relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command) in the honorary rank of Captain, 30th April, 1956. To be Captain (provisionally) and Temporary Major, 13th June, 1956: 3/157159 Nicholas Talbot Hamilton.

Central Command.

Royal Australian Army Medical Corps (Medical).—To be Captain (provisionally), 22nd March, 1956: 4/35506 Victor Horvat.

Western Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 5/26532 Captain E. F. Hayward is confirmed. To be Major, 30th July, 1955: 5/21578 Captain (Temporary Major) C. Stuart, M.C. To be Captain (provisionally), 15th May, 1956: 3/129248 Cedric Rupert Naylor.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Southern Command.—To be Honorary Captains: Kevin James Green, 8th April, 1956, and Roger Anthony Williams, 19th May, 1956.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Barrymore Walters (037263) is appointed to a short-service commission, on probation, 26th March, 1956, with the rank of Pilot Officer (Student).

The resignation of Flight Lieutenant P. J. Bayliss (036611) is accepted, 15th June, 1956.

Active Citizen Air Force: Medical Branch.

No. 21 (City of Melbourne) Squadron.—Flight Lieutenant B. W. Costello (033455) is transferred to the Reserve, 30th November, 1955.

Air Force Reserve: Medical Branch.

Flight Lieutenant (Temporary Wing Commander) D. R. Gawler, M.C. (1183), is placed on the Retired List, 30th September, 1954.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 11, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(2)	3(2)	3	..	..	..	1	..	9
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	1	..	4	..	..	..	..	..	5
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1(1)	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1	15(13)	9(1)	..	1	..	2	..	28
Diphtheria	..	1(1)	1	..	1	..	..	..	3
Dysentery (Bacillary)	..	..	..	3(3)	..	..	3	..	6
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	47(14)	38(14)	..	10(9)	2(1)	3	2	..	102
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	..	..	..	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	3	..	3
Meningococcal Infection	1(1)	1(1)	2	..	..	3(1)	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(3)	..	1	3(2)	1	1	..	..	10
Puerperal Fever	..	..	8	..	..	..	..	..	8
Rubella	..	4(4)	..	5(4)	4(4)	..	1	..	14
Salmonella Infection	..	..	..	3(2)	..	..	..	..	3
Scarlet Fever	9(4)	11(5)	8(3)	5(2)	2(2)	..	..	..	35
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1(1)	..	..	..	1
Trachoma	..	..	..	..	1	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	24(14)	13(9)	7(5)	7(5)	6(5)	3(2)	1	..	61
Typhoid Fever	2(2)	..	..	..	..	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1(1)	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End at Kempsey.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at the Nurses' Home, Macleay District Hospital, Kempsey, in conjunction with the Eastern District Medical Association, on Saturday and Sunday, September 15 and 16, 1956. The programme is as follows:

Saturday, September 15: 2.15 p.m., registration; 2.30 p.m., "Modern Concepts of the Medical Management of Liver Disease", Dr. Kenneth B. Noad; 4.15 p.m., "Modern Concepts of the Surgical Management of Liver Disease", Dr. Alan Sharp.

Sunday, September 16: 9 a.m., annual general meeting of the Eastern District Medical Association; 11.30 a.m., "Some Medical Aspects of Peripheral Vascular Disease", Dr. Kenneth B. Noad; 2 p.m., "Some Surgical Aspects of Peripheral Vascular Disease", Dr. Alan Sharp.

The fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. Angus McNeil, Honorary Secretary, Eastern District Medical Association, 89 River Street, West Kempsey (telephone Kempsey 194), particularly mentioning whether hotel accommodation is required. Members are asked to note that the annual subscription to the Eastern District Medical Association is now due.

Notice.

VICTORIAN MENTAL HEALTH EXHIBITION.

THE Mental Hygiene Authority of Victoria will hold the first Victorian Mental Health Exhibition at the Melbourne Town Hall on September 18 and 19, 1956. The exhibition will be opened by the Honourable E. P. Cameron, M.L.C., Minister for Health, at 2 p.m. on Tuesday, September 18. The displays will illustrate the following aspects of the work carried out by the Mental Hygiene Authority: mental hospital services, out-patient clinics, treatment and research, occupational therapy, psychiatric art, psychiatric nursing, community education *et cetera*.

FAIRFAX READING MEMORIAL PRIZE.

THE Fairfax Reading Memorial Prize of the Dental Alumni Society of the University of Sydney for the year 1956 has been awarded to Professor A. J. Arnott, Dean of the Faculty of Dentistry, University of Sydney. The Fairfax Reading Memorial Prize is awarded biennially, and it has on this occasion been awarded to Professor Arnott for his outstanding contribution to the development in the teaching and training of dentists and the expansion of dental services in New South Wales.

THE AUSTRALASIAN ASSOCIATION OF PSYCHIATRISTS.

THE New South Wales Branch of the Australasian Association of Psychiatrists will hold a clinical meeting on Thursday, October 11, 1956, at 7.30 p.m., at Broughton Hall Psychiatric Clinic, Wharf Road, Leichhardt. All members of the medical profession are invited to attend.

Medical Appointments.

Dr. J. F. J. Cade has been appointed Superintendent of the Receiving Houses, Pleasant View and Royal Park, Victoria.

Dr. R. R. Webb has been appointed Deputy Superintendent of the Receiving Houses, Pleasant View and Royal Park, Victoria.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Nevasky, Zygmunt-Stanley, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1955 (subject to Section 17 (*a*)), 295 Edgecliff Road, Woollahra, New South Wales.

Deaths.

THE following death has been announced:

BOTTOMLEY.—Edward Eric Bottomley, on July 29, 1956, at Hampton, Victoria.

Diary for the Month.

SEPT. 5.—Western Australian Branch, B.M.A.: Branch Council.
SEPT. 7.—Queensland Branch, B.M.A.: General Meeting.
SEPT. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 14.—Tasmanian Branch, B.M.A.: Branch Council.
SEPT. 14.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 30 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.